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Changes in hematological and other laboratory parameters in COVID-19 infection

Prakas Kumar Mandal[1], Rishu Vidhatri[1]

[1]Department of Hematology, Nil Ratan Sircar Medical College, Kolkata, West Bengal, India.

***Corresponding author:**
Dr. Rishu Vidhatri,
Department of Hematology, Nil Ratan Sircar Medical College, Kolkata, West Bengal, Kolkata, India.

rishuvidhatrib19@gmail.com

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ABSTRACT

Severe acute respiratory syndrome-CoV-2 was declared as a pandemic by the World Health Organization in March 2020. The virus belongs to the family Coronaviridae and causes infection of varying severity ranging from mild respiratory tract infection to severe pneumonia or acute respiratory disease syndrome. Several laboratory parameters are deranged in COVID-19 infection. The gold standard of diagnosis of COVID-19 infection is polymerase chain reaction (PCR) from the nasopharyngeal and oropharyngeal swab. However, at remote places, where PCR reports are made available to patients after a time gap laboratory parameters may guide the treating physician regarding diagnosis, disease severity, and prognosis.

Keywords: COVID-19 infection, Laboratory parameters, Disease severity

INTRODUCTION

Coronavirus belongs to the family Coronaviridae. It has caused two major pandemics in the past 20 years, one in 2002 severe acute respiratory syndrome (SARS-CoV) and the other in 2012 Middle East respiratory syndrome-CoV.[1] This time the pandemic is caused by SARS-CoV-2 which is the seventh known coronavirus to infect humans. The novel virus infection was first reported in Wuhan, China, in December 2019 and then rapidly spread worldwide. SARS-CoV-2 was declared as an emergency on January 30, 2020, by the World Health Organization (WHO) and shortly on March 11 was declared a pandemic by the WHO.[2] It has ravaged the whole world over the past 2 years and recently by new mutant more virulent strains. In India, the first case was detected on January 30, 2020, in a student who returned from Wuhan University to Thrissur, Kerala.[3] Since then, millions of infected cases have been reported from India. The clinical manifestation of the disease varies from asymptomatic cases to severe cases with multiorgan dysfunction. The key to controlling the pandemic is early diagnosis, containment, disease stratification, and proper management.

PATHOPHYSIOLOGY

The SARS-CoV-2 virus is an enveloped single-stranded (ss) ribonucleic acid (RNA) virus and belongs to the family Coronaviridae.[4] Bats are believed to be the animal reservoir of the virus.[5] The virus is transmitted from person to person through a respiratory droplet. The coronavirus is made of four proteins, the spike proteins (S), membrane (M), envelop

(E), and nucleocapsid (N) proteins.[6] These S proteins bind to the angiotensin-converting enzyme receptor 2 present on pulmonary epithelial cells of the host and multiple copies of ssRNAs are formed by enzyme RNA-dependent RNA polymerase. The viral RNA integrates with the host ribosome and translates to form a complete virion which is released from the pneumocyte. The injured pneumocyte releases various inflammatory cytokines, leading to disease complications. The incubation period of COVID-19 is 5–6 days but can be up to 14 days. The virus can infect patients of any age, most seen in 40–70 years, and present with fever, body aches, breathlessness, dry cough, abdominal pain, vomiting, and loose stools. According to disease severity, it can be asymptomatic, mild, moderate, or severe disease.[7,8]

CURRENT DIAGNOSTICS OF COVID-19

Confirmatory test for COVID-19 infection is molecular identification of SARS-CoV-2 using nucleic acid amplification tests such as the reverse transcriptase-quantitative polymerase chain reaction (RT-PCR) or viral gene sequencing.[9] Samples used are nasopharyngeal, oropharyngeal swab, sputum, or bronchoalveolar fluid. Rapid diagnosis of infection can also be done using rapid antigen test kits. However, in the developing countries, where laboratories are not well equipped and there is a lack of trained technicians to perform massive molecular tests in this pandemic which may lead to delay in results, during which clinical assessment is crucial for patient management. During this period, the patient history, hematological and biochemical laboratory parameters, and imaging in selected cases aid in the clinical confirmation of diagnosis. Chest X-ray is typically inconclusive in the early stages of the disease; as the infection progresses bilateral multifocal alveolar opacities are observed, which can even be associated with pleural effusion. High-resolution computed tomography is extremely sensitive even in the early phase of the disease and shows multifocal bilateral "ground-glass" areas associated with consolidation and patchy peripheral distribution, with greater involvement of the lower lobes.[10]

HEMATOLOGICAL INDICES

Hematological indices vary in COVID-19 patients according to disease severity. In the developing countries with resource constraint settings where molecular reports may take several days to come, these parameters at least may give some clue to treating physicians toward diagnosis and disease severity. In the COVID-19 pandemic, the clinical and laboratory findings may help the treating physician to narrow down the differential diagnosis, facilitate early isolation of patients, and provide symptomatic treatment based on the laboratory parameters which are the treatment option available for this novel viral infection. Published literature has shown that there are leukopenia, lymphopenia, high neutrophil-to-lymphocyte ratio, and thrombocytopenia.[11-26] [Table 1] shows alteration in hematological parameters published in various studies. Thrombocytopenia with significant bleeding is unusual. Hemoglobin remains normal in most cases until there is a severe infection. Whether these alterations in hematological parameters are associated with morbidity and mortality in COVID-19 infection is not well established and there is conflicting evidence for the same.

In severe infection, Tang *et al.*[26] found that thrombocytopenia negatively correlated with 28-day mortality. Our experience with this novel viral infection is new and published literature has shown that there is an increase in leukocyte, high neutrophil-to-lymphocyte ratio, lymphocytopenia, and thrombocytopenia with the progression of the disease.[17,18] Lymphocytopenia is a common abnormal laboratory parameter found in COVID-19 infection and may give a clue about disease prognosis in the initial phase of infection. Out of 1099 patients studied by Guan *et al.*,[28] 83.2% of patients had lymphocytopenia, and leukocytosis was associated with severe disease. Qin *et al.*[17] studied 452 patients of which 286 had a severe infection and found a significant ($P < 0.001$) higher leukocytosis, neutrophilia, high neutrophil-to-lymphocyte ratio, and lymphocytopenia in the severe group when compared to the non-severe group. Huang *et al.*[14] and Wang *et al.*[20] found a significant association between lymphopenia and intensive care unit (ICU) admission. Like the above-mentioned studies, Fan *et al.*[13] also found that on admission, low lymphocyte count was significantly ($P < 0.001$) associated with ICU admissions. While the above discussed published literature found an association between hematological parameters and disease severity or prognosis, other studies reveal conflicting results. Fan *et al.*[13] showed that there was no association between thrombocytopenia at either admission or during hospitalization and ICU admission. Wu *et al.*[21] found a non-significant difference in platelet count between survivors and non-survivors complicated by acute respiratory distress syndrome. Young *et al.*[8] found that platelet count was not different in patients requiring oxygen support and those who did not, but the statistical comparison was not done. Wang *et al.*[20] and Wu *et al.*[21] also found that there was a non-significant difference in platelet count between hospitalized patients infected with SARS-CoV-2 in ICU and non-ICU patients. Liu *et al.*[15] found a non-significant difference in platelet, leukocyte, and lymphocyte count in patients with progressive and stable SARS-CoV2 infection.

THROMBOTIC BIOMARKERS

Apart from hematological indices, thrombotic biomarkers are also frequently abnormal in SARS-CoV-2 infection.

Table 1: Changes in hematological indices in COVID-19 infection.

References	Sample size	Platelet	WBC	Hemoglobin
Ding *et al.*[11]	*n*=72 15 (20.8%) severe 57 (79.2%) non-severe	Thrombocytopenia Severe – 26.7% Non-severe – 10.5% (*P*=0.108)	Lymphopenia in severe 73.3% and 49.1% of non-severe cases (*P*=0.09) –Leukocyte (*P*=0.05), Neutrophil: lymphocyte ratio (*P*=0.002) was increased Lymphocyte: monocyte ratio was decreased (*P*=0.102) in severe patients	Hb Severe – 13.9 Non-severe – 14 (*P*=0.51)
Chen *et al.*[12]	Single-center study on RT-PCR confirmed cases (*n*=99)	Thrombocytopenia 12% Thrombocytosis – 4%	Leucopenia 9% Leukocytosis 24% Neutrophilia 38% Lymphopenia 35%	Decrease in hemoglobin – 51%
Fan *et al.*[13]	*n*=67 58 – non-ICU 9 – ICU	Thrombocytopenia ICU – 21% Non-ICU – 11% (*P*=0.67)	Leukopenia 29.2% (*P*=0.87) Lymphopenia 36.9% (*P*=0.0002)	Hb slightly decreased in ICU patients. Difference non-significant (*P*=0.07)
Huang *et al.*[14]	*n*=41 ICU=13 Non-ICU=28	Thrombocytopenia ICU – 8% Non-ICU – 4% (*P*=0.45)	Leucopenia ICU – 8% Non-ICU – 33% Leukocytosis ICU – 54% Non-ICU – 19% (*P*=0.04) Lymphopenia ICU – 85% Non-ICU – 54% (*P*=0.04) Median absolute neutrophil count ICU – 10·6×10^9/L (IQR 5.0–11.8) non-ICU – 4.4×10^9 (IQR 2·0–6·1) (*P*=0·00069)	Hb slightly decreased in ICU patients. (*P*=0.2)
Liu *et al.*[15]	*n*=78 Disease progression – 11 Disease improvement/ stabilized 67	Disease progression –143.9±64.81×109/L Disease stabilized –173.2±55.37×109/L (*P*=0.116)	TLC in disease progression – 6.08±2.56 x109/L Stabilization groups –5.18±1.63×109/L (*P*=0.294). Lymphocytes are slightly lower in disease progression Group (*P*=0.075)	
Qian *et al.*[16]	*n*=91	Severe – 152 (127–208)×10^9/L Non-severe – 198 (144–248) ×10^9/L *P*=0.51	TLC Severe – 5.23 (4.74–6.8)×10^9/L Non-severe – 4.97 (4.02–5.65)×10^9/L (*P*=0.010) Lymphocyte Severe – 0.9 (0.7–1.3)×10^9/L Non-severe – 1.4 (1.05–1.75)×10^9/L (*P*=0.027) Neutrophil severe – 3.32 (3–5.82) Non-severe – 2.8 (2.18–3.49) (*P*=0.0004)	Hb severe – 130 (118–142) Non-severe – 135 (126–147) (*P*=0.27)
Qin *et al.*[17]	*n*=452 Non-severe – 166 Severe – 286	Thrombocytopenia more significant in severe infection (*P*=0.001)	Severe cases had higher leukocyte (5.6 vs. 4.9×10^9; *P*<0.001), neutrophil (4.3 vs. 3.2×10^9; *P*<0.001) counts, lower lymphocytes count (0.8 vs. 1.0×10^9; *P*<0.001), higher neutrophil-to-lymphocyte ratio (5.5 vs. 3.2; *P*<0.001), lower percentages of monocytes (6.6 vs. 8.4%; *P*<0.001), eosinophils (0.0 vs. 0.2%; *P*<0.001), and basophils (0.1 vs. 0.2%; *P*=0.015)	
Ruan *et al.*[18]	*n*=150 Survivor – 82 Non-survivor – 68	Platelet count significantly lower in non-survivors than in survivors	Leukocytosis and lymphopenia were significantly more in non-survivors	

(Contd...)

Table 1: *(Continued)*.

References	Sample size	Platelet	WBC	Hemoglobin
Wang *et al.*[19]	69 cases SpO2>90% *n*=55 SpO2<90% *n*=14	Normal in both groups	Leukopenia severe –21% Non-severe – 62% (*P*=0.007) Leukocytosis Severe – 7% Non-severe Lymphocytopenia Severe – 79% Non-severe – 32% (*P*=0.002)	Normal
Wang *et al.*[20]	*n*=138 ICU – 36 Non-ICU – 102	Median platelet count was slightly lower in ICU patients than in non-ICU patients (*P*=0.78)	Higher TLC ICU –6.6 (3.6–9.8)×10^9/L Non-ICU – 4.3 (3.3–5.4)×10^9/L; *P*=0.003, higher neutrophil count in ICU patients than in non-ICU patients (*P*<0.001) Lymphocytopenia more in ICU patients difference (non-significant)	
Wu *et al.*[21]	*n*=201 84 (41.8%) developed ARDS	Thrombocytopenia 18.8%	Leukocytosis 23.4% Lymphopenia 64.0% Neutrophilia 34.5%	
Yang *et al.*[22]	52 Critically ill 20 Survivors 32 Non-survivors	Median platelet count was normal in survivors and non-survivors	Lymphocytopenia 85% No association between survivors and non-survivors	
Zachariah *et al.*[23]	*n*=50 Non-severe – 41 Severe – 9		Leukocytosis (*P*=0.003) Severe – 23.2% Non-severe – 4.9% Leukopenia (*P*=0.16) Severe – 16.1% Non-severe – 22% Lymphopenia 72% Non-significant between severe and non-severe (*P*=0.160)	
Zhang *et al.*[24]	*n*=140 Non-severe – 58 Severe – 82		Leukopenia – 19.6% (*P*=0.001) Lymphocytopenia –75.4% (*P*=0.001) Eosinophil lowered 51.9% (*P*=0.06) Significantly difference between severe and non-severe	
Zhou *et al.*[25]	*n*=191 Survivors – 137 Non-survivors – 54	Thrombocytopenia 7% More non-survivors showed thrombocytopenia than survivors. (*P*<0.0001)	Lymphocytopenia 40% Leukocytopenia 17% Significant difference between non-survivors and survivors (*P*<0.0001)	

Hb: Hemoglobin, TLC: Total leukocyte count, ICU: Intensive care unit, ARDS: Acute respiratory distress syndrome, and RT-PCR: Reverse transcriptase polymerase chain reaction

Coagulation parameters abnormalities found in COVID-19 infection published in the literature are summarized in Table 2. A D-dimer level may help in the prognostication of disease and available literature has shown an association with disease severity.[14,19,21] Prothrombin time (PT) and activated partial thromboplastin time (a PTT) were more in severe SARS-CoV-2 infection; however, the difference was non-significant.[14,22] Han *et al.*[27] did a study to assess abnormalities of coagulation parameters in COVID-19 infection and found that D-dimer, fibrin/fibrinogen degradation products (FDPs), and fibrinogen were increased in all patients, and D-dimer and FDP values were higher in patients with severe infections compared to those with mild infection. Tang *et al.*[26]

Table 2: Changes in coagulation profile in COVID-19 infection.

Reference	PT (s)	aPTT (s)	D Dimer (mg/L)
Chen *et al.*[12]	Increased 5% Decreased 30%	Increased 6% Decreased 16%	Increased 36%
Huang *et al.*[14]	Median ICU – 12.2 s (IQR 11.2–13.4) Non-ICU – 10.7 (IQR 9.8–12.1) (*P*=0.012)	Median ICU – 26.2 s (IQR 22.5-33.9) Non-ICU – 27.7 s (IQR 24.8–34.1) (*P*=0.57)	Median ICU – 2.4 (0.6–14.4) Non-ICU – 0.5 (0.3–0.8) (*P*=0.0042)
Liu *et al.*[15]			D-dimer more in disease progression however difference non-significant (*P*=0.501)
Qian *et al.*[16]			Severe – 450 (160–485) ng/ml Non-severe – 300 (106–400) ng/ml (*P*=0.591)
Wang *et al.*[20]	Raised in both arms (non-significant)	Normal	Significant increase in ICU group (*P*<0.001)
Wu *et al.*[21]	Prolonged 2.1%	Prolonged 9.7%	Increased 23.3%
Zachariah *et al.*[23]			Non-severe – 0.2 (0.1–0.3) μg/mL Severe – 0.4 (0.2–2.4) μg/mL (*P*≤0.001)

PT: Prothrombin time; aPTT: activated partial thromboplastin time, ICU: Intensive care unit

found significantly increased D-dimer and FDP levels, and prolonged PT and aPTT in non-survivors compared to survivors. Zhou *et al.*[25] found that D-dimer greater than 1 μg/mL on admission was predictive of in-hospital mortality (P = 0.0033). Wang *et al.*[20] found a significant difference between D-dimer values in ICU as compared to non-ICU patients while there was a non-significant increase in PT in COVID patients and a PTT was normal and there was no significant difference between ICU and non-ICU patients.

BIOCHEMICAL PARAMETERS

COVID-19 infection causes activation of the immune system and release of various cytokines which cause injury to vital organs such as the lung, kidney, heart, and liver, resulting in derangement of many biochemical parameters. Biochemical parameters deranged in SARS-CoV-2 infection are enlisted in Table 3. Hypoalbuminemia was significantly more common in severe disease as reported in various studies.[14,15,18,19] Increased lactate dehydrogenase (LDH) level was significantly associated with severe disease.[19-21] Liver enzymes were increased in patients with SARS-CoV-2 infection although the rise was marginal.[12,14,20,22] Many studies have reported aspartate transaminase to be significantly raised in severe cases compared to non-severe.[19,21] Total bilirubin was marginally more in patients with severe disease.[12,14,19,22]

Blood urea nitrogen and creatinine levels were increased more in severe infection.[21,22]

IMMUNOLOGICAL PARAMETERS

The most common immunological abnormality in COVID-19 infection was interleukin 6 (IL-6), erythrocyte sedimentation rate, ferritin, and C-reactive protein (CRP). Immunological parameters deranged in COVID-19 infection are summarized in Table 4. Many studies have documented that CRP is significantly elevated in severe disease as compared to non-severe disease and is an important marker of disease progression and response to treatment.[15,17-20] Ferritin was increased more in patients who were severely infected compared to those with non-severe infection.[17,18] Procalcitonin is not commonly increased in COVID-19 patients and is often raised in patients with a secondary bacterial infection. Huang *et al.*[14] did a study on 41 patients and found that procalcitonin levels were raised in four patients and all four had a secondary bacterial infection. Qin *et al.*,[17] in a study on 452 patients, found that procalcitonin was significantly elevated in ICU admitted patients who were severely infected with COVID-19 infection than in non-ICU patients (0.1 vs. 0.05 ng/mL; P < 0.001), and many inflammatory cytokines were significantly raised in severe infection than the non-severe ones, including IL-2R, IL-6, IL-8, IL-10, and tumor necrosis factor-alpha. Immunoglobulins such as IgA, IgG, and IgM and complement proteins (C3 and C4) were not affected or they were within the normal range. T lymphocytes are significantly decreased in severe COVID-19 infection as compared to non-severe. Both helper T lymphocytes and suppressor T lymphocytes were below normal in all patients and helper T cells were

Table 3: Changes in biochemical parameters in COVID-19 infection.

Reference	Albumin (gm/L)	ALT (IU/L)	AST (IU/L)	Bilirubin (mg/L)	BUN	Creatinine	LDH (IU/L)
Chen *et al.*[12]	Decreased 98%	Increase d 98%	Increase d 35%	Increased 18%	Increased 6% Decrease d 17%	Increase d 3% Decreased 21%	Increased in 76%
Fan *et al.*[13]							Increased in 43.6% ICU patients (*P*=0.005)
Huang *et al.*[14]	Median ICU – 27.9 (26.3–30.9) Non-ICU – 34.7 (30.2–36.5) (*P*=0.00066)	Median ICU – 49 (29–115) Non-ICU – 27 (19.5–40) (*P*=0.038)	<40 ICU – 38% Non-ICU – 75% (*P*=0.025)	ICU – 14.0 (11.9–32.9) Non-ICU – 10.8 (9.4–12.3) (*P*=0.011)			>245 ICU (92%) Non-ICU – 635 (*P*=0.03)
Liu *et al.*[15]	Progression group – 36.62±6.60 Stabilization group – 41.27±4.55 (*P*=0.006)	No significant difference (*P*=0.77)	No significant difference (*P*=0.78)				
Qian GQ16	Severe – 38.55 (36.33–39.25) g/l Non-severe – 40.2 (38–42.4) g/l (*P*=0.133)	Severe – 19.9 (14–26) U/l Non-severe – 18 (13–29) U/l (*P*=0.75)	Severe – 27 (23.75–27) U/l Non-severe – 21 (17–29) U/l (*P*=0.89)		Severe – 5.19 (4.66–6.14) mmol/L Non-severe – 3.83 (3.25–4.4) mmol/L (*P*=0.0001)	Severe – 81.5 (70.75–90.5) umol/l Non-severe – 66 (57–76) umol/l (*P*=0.03)	
Ruan *et al.*[18]	Significantly lower in non-survivors				Significantly high in non survivors		
Wan *et al.*[19]		Slightly more in the severe group (*P*=0.11)	More in the severe group (*P*<0.03)				Significantly higher in severe cases (*P*=0.001)
Wang *et al.*[20]		Normal	Increase in ICU (*P*<0.001)	Normal	Increased in ICU (*P*<0.001)	More in ICU patients (*P*=0.04)	Increase in ICU (*P*<0.001)
Wu *et al.*[21]	Decreased 98.5%	Increase d 21.7%	Increase d 29.8%	Increased 5.1%	Increased 4.5%	Increased 4.5%	Raised 68.2%

ALT: Alanine transaminase, AST: Aspartate transaminase, BUN: Blood urea creatinine, LDH: Lactate dehydrogenase; and ICU: Intensive care unit

Table 4: Changes in immunological parameters in COVID-19 infection.

Reference	Procalcitoni n	IL-6	ESR	Ferritin	CRP (mg/L)
Chen *et al.*[12] Huang *et al.*[14]	Increased 6% >0.5 ng/ml ICU-25% Non-ICU-Nil	Increased 52%	Increased 85%	Increased 63%	Increased 86%
Liu *et al.*[15]	Non-significant difference between progressive and stable disease (*P*=0.195)		Non-significant difference between progressive and stable disease (*P*=0.794)		Progression group versus improvement/stabilization group (38.9 [14.3, 64.8] vs. 10.6 [1.9, 33.1] mg/L, U=1.315, *P*=0.024)
Qian *et al.*[16]	Severe – 0 (0) Non-severe – 0.03 (0–0.04) ng/m (*P*=0.003)				Severe – 30.63 (12.5–103.4) mg/l Non-severe – 5.98 (1.4–11.3) mg/l (*P*≤0.0001)

(Contd...)

Table 4: *(Continued).*

Reference	Procalcitoni n	IL-6	ESR	Ferritin	CRP (mg/L)
Qian *et al.*[16]	Increased in severe infection (0.1 vs. 0.05 ng/mL; *P*<0.001)	IL-6, 8, and 10 Significantly more in severe cases	Non-significant difference between severe and non-severe	Increased in severe (800.4 vs. 523.7 ng/ml; *P*<0.001)	
Ruan *et al.*[18]		Significantly higher in non-survivors		Significantly higher in non-survivors	Significantly higher in non-survivors
Wang *et al.*[19]	Increased in severe cases (*P*=0.78)				Significantly higher in severe infection (*P*=0.001)
Wang *et al.*[20]	Procalcitonin ≥0.05 ng/ml severe – 75% non-severe – 21.6% (*P*≤0.001)				
Wu *et al.*[21]	Raised in 85.6%	Raised in 48.8%	Raised in 93.8%	Raised in 78.5%	Raised in 85.6%
Zachariah *et al.*[23]	Severe – 0.1 (0.06–0.3) ng/mL Non-severe-0.05 (0.03–0.1) ng/mL				Severe – 47.6 (20.6–87.1) mg/l Non-severe – 28.7 (9.5–52.1) mg/l *P*=0.001

IL-6: Interleukin 6, ESR: Erythrocyte sedimentation rate, CRP: C-reactive protein, and ICU: Intensive care unit

significantly decreased in the severe group. Naïve helper T cells were increased in severe infection, and the number of memory helper T cells decreased in severe cases.[17]

CONCLUSION

Several laboratory parameters are affected in COVID-19 infection. Total leukocyte count, lymphocytopenia, thrombocytopenia, CRP, D-dimer, albumin, and LDH have been shown in published literature to have prognostic implications. The above-mentioned studies highlight the necessity and importance of laboratory tests to be done in COVID-19 infection. The gold standard diagnostic test for COVID-19 is RT-PCR.

Test for COVID-19 is RT-PCR, however, in resource constraint countries, where molecular test results are made available after a few days, these laboratory parameters may at least give a minimum diagnostic and prognostic clue to treating physicians.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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