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Review Article

Hematological and Immunological Laboratory findings among COVID 19 Patients

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ABSTRACT

Background: Every day, more and more countries throughout the world reported a rise in the prevalence of COVID-19 infections. Finding clinical and laboratory indicators of worsening disease severity and mortality are of the utmost importance as this epidemic continues to spread. Recovery from a COVID-19 infection does not eliminate the effects on many hematological and immunological laboratory parameters. In this study we aimed to assess Hematological and Immunological Laboratory findings among COVID 19 patients and if there is possible relationship between these parameters with Covid-19 disease severity.

Conclusion: In the absence of evidence from a real-time polymerase chain reaction or antibodies, a thorough familiarity with the laboratory findings and hematological abnormalities linked to SARS-CoV-2 infection might aid in raising suspicion of illness. Rapid screening, diagnosis, prognosis, and treatment of COVID-19 patients could be aided by blood counts in addition to morphological alterations in peripheral blood. Both the diagnosis and the prognosis depend on the thrombocytopenia count. Infection with COVID-19 is indicated by low leukocyte and neutrophil counts, but the opposite is true for progressive infection: increased levels.

Keywords: Hematological, Immunological, Laboratory findings, Recovery, COVID 19 infection.

INTRODUCTION

In terms of health, economics, and lifestyle, the COVID-19 pandemic, which was triggered by SARS-CoV-2, has been a terrible menace to human society. Despite the fact that the virus often infects the respiratory tract and lungs first, it is now recognized to have a detrimental impact on nearly every major organ in the body, which can cause catastrophic systemic failure in certain individuals [1].

There is presently no cure for this illness, which is quite unfortunate. Premature death and increased morbidity and mortality are mostly caused by pre-existing medical diseases or comorbidities, such age. There is an increased risk of harm to the immunological, respiratory, cardiovascular, musculoskeletal, and brain systems as a result of the immobility caused by hospitalization, bed rest, and physical inactivity caused by prolonged quarantine and social isolation [2].

Complete Blood Count (CBC):

Patients diagnosed with COVID-19 have changes in their blood counts that are associated with the disease's progression and severity. Leukocyte parameters in COVID-19 infection; The peripheral blood smear of an infected patient shows several morphological abnormalities, suggesting that SARS-CoV-2 inflicts a direct cytopathic damage on cells. A characteristic of SARS-CoV-2 infection, lymphopenia is present in almost all symptomatic individuals, albeit to varying degrees. Evidence also suggests a correlation between the degree of

lymphocyte count drop and the severity of the disease [3].

Infection with COVID-19 is also characterized by a low eosinophil level. In a patient experiencing symptoms, a low eosinophil count in conjunction with lymphopenia strongly suggests infection. Researchers have discovered that a high neutrophil count in COVID-19 patients portends a bad outcome. A high neutrophil-to-lymphocyte ratio (NLR) is indicative of potential complications when combined with a low lymphocyte count [4].Lympopenia is the most well-known haematological aberration in COVID-19 patients; it occurs in as many as 85% of severe cases and is associated with prognosis. The majority of published series report lymphopenia, which is characterized by a total lymphocyte count below 1.0 $x10^9$/L, and is often thought of as an inadequate immune response to a viral infection [5].

The pathogenic mechanism of COVID-19 includes a hyperinflammatory state and cytokine storm, which are correlated with neutrophilia, with the exception of individuals with bacterial infections or superinfections. The number of neutrophils in the bloodstream increases slowly but steadily as COVID-19 advances. Patients with severe COVID-19 infection had considerably higher levels of leukocytes and neutrophils compared to those with less severe infections. Additionally, severe COVID-19 patients had higher leukocyte and neutrophil counts as the disease progressed [6].

Studies have shown that the neutrophil-lymphocyte ratio (NLR) and platelet-lymphocyte ratio (PLR) can be helpful in the diagnosis, monitoring, and evaluation of a range of systemic inflammatory processes, including cholangiocarcinoma, ischemic heart disease, acute pancreatitis, and malignant tumors [7].

Red blood cells and hemoglobin:

It was shown that SARS-CoV-2 infection significantly affects the protein and lipid balance of the structural membrane of red blood cells (RBCs). Red blood cells (RBCs) infected with COVID-19 exhibited elevated amounts of glycolytic intermediates, protein oxidation, and membrane protein disintegration. That is why COVID-19 affects the red cell membrane and the haemoglobin oxygen affinity—two crucial processes. The ability of red blood cells (RBCs) from COVID-19 patients to carry and deliver oxygen may be impaired because they are unable to adapt to changes in ambient hemoglobin oxygen saturation on their way from the lungs to the bloodstream [8].

Platelets:

An integral part of many disease severity classification systems, including the one for multiorgan dysfunction syndrome, is the platelet (PLT) count. Thrombocytopenia is an indicator of consumption coagulopathy and correlates with illness severity in COVID-19 infection. There was an association between a low PLT count and increased mortality and severe COVID-19 disease [9].

Coagulation Profile (PT, Ptt , Fibrinogen and D-Dimer):

Patients with COVID-19 have been found to experience coagulopathy in multiple investigations. Other coagulopathies, such DIC, were not present. In comparison to controls, COVID-19 patients exhibited elevated D-dimer levels (> 1,000 ng/mL) and longer prothrombin times. It may be useful as a prognostic indicator for determining which individuals should receive early treatment intervention because levels were higher in patients with severe disease compared to non-severe disease [10].

In COVID-19 patients, in-hospital death may be predicted by an admission D-dimer level more than2.0 µg/mL. The COVID-19 virus is associated with three to six times the usual incidence of thrombosis. Particularly in the acute phase of DIC, increased PT, INR, and aPTT levels are common. Unlike traditional sepsis, COVID-19 patients typically have normal PT and aPTT; in fact, only 6% of patients with the virus experience prolonged PT and/or aPTT [11].In situations of late-stage or severe DIC, fibrinogen levels, a very specific diagnostic sign, may be low. A mean of 4.55 g/L of hyperfibrinogenemia was seen in the majority of COVID-19 patients [12].

Liver and Kidney Function Tests:

Numerous studies have shown that individuals with severe COVID-19 disease often have aberrant results on laboratory tests. Most patients had elevated ALT and AST levels; 7590 U/L for ALT and 1445 U/L for AST, respectively. In addition, 36.2% of COVID-19 patients had an elevated gamma glutamyltransferase (GGT) level of 55 U/L, 9.6% had an alkaline phosphatase (ALP) level of 150 U/L, and 10.6% had total bilirubin >1.2 mg/dl [13].

In patients admitted to the hospital with COVID-19, acute kidney damage (AKI) is prevalent; it has been recorded in 24% to 57% of COVID-19 hospitalizations and 61% to 78% of critical care unit admissions. Patients infected with COVID-19 are

more likely to get severe acute kidney injury (AKI), have higher dialysis needs, and have a slower renal recovery while hospitalised, all of which may raise their risk of developing chronic kidney disease (CKD) or making their current CKD worse [14].

Serum LDH:

Several biomarkers are being studied to see whether they might help predict how COVID-19 patients will do in the future. One such biomarker of relevance is lactate dehydrogenase (LDH), particularly because higher LDH levels have previously been linked to poorer outcomes in individuals with other viral infections. Preliminary data from COVID-19 participants reveals notable variations in LDH levels between those with and without severe illness [15].

Inflammatory Parameters:

1- Erythrocyte Sedimentation Rate (ESR):

In comparison to patients without severe sickness, those with critical illness had a higher ESR. Research on the connection between COVID-19 and ESR is limited. Researchers were unable to find any alternative explanation for the high ESR levels because ESR stayed higher for an extended period. Studies examining illness severity and prognostic markers have used ESR as one of several metrics for evaluation [16].

2- C-Reactive Protein (CRP):

While C-reactive protein has traditionally been used to indicate acute phase inflammation, in the COVID-19 pandemic, it is now associated with tissue damage and a poor prognosis. Here, elevated C-reactive protein levels in the early stages of COVID-19 have been linked to lung damage and disease severity; in fact, elevated C-reactive protein levels are present before lung lesions emerge, providing C-reactive protein predictive values of disease severity [17].

3- Serum Ferritin:

By its direct immunosuppressive and pro-inflammatory effects, ferritin contributes to the cytokine storm and plays a crucial role in immunological dysregulation, particularly in cases of severe hyperferritinemia. There is evidence that cytokine storm syndrome is associated with COVID-19 lethal outcomes; hence, it has been postulated that the severity of the disease is dependent on this condition [4].

Serum Electrolytes:

Multiple clinical problems seen by COVID-19 patients might lead to electrolyte abnormalities. Hyponatremia in adults with COVID-19 may be associated with the increased secretion of antidiuretic hormone (ADH) in reaction to a decrease in blood volume caused by fluid loss from the gastrointestinal tract. Because the incidence of hypokalemia in COVID-19 patient is mostly unknown. Research on the pathophysiology of COVID-19 has shown that potassium loss in the urine is the main cause of hypokalemia [18].

Immunoglobulins:

The current gold standard for COVID-19 diagnosis is the detection of SARS-CoV-2 RNAs. Nevertheless, quick and accurate serological diagnostic tools are desperately needed to screen for SARS-CoV-2 infections, even in patients without obvious symptoms. Emerging research overwhelmingly outlined serological assays that detect IgM and IgG antibodies unique to SARS-CoV-2. Despite a small number of publications reporting SARS-CoV-2-specific IgA in serum, there is a dearth of studies analyzing IgA levels in a larger sample of COVID-19 patients [19].

The only way to reliably diagnose a case of COVID-19, especially at an early stage of illness, and to find out if a patient is infectious to others is to use molecular test methods. It is possible to tell if a patient is contagious by looking for viral genetic material in their nasal, oral, and respiratory tracts. With the ability to establish viral presence up to two days before symptoms appear, these tests are probably most useful when administered early in the course of an infection. Molecular tests can shorten the diagnosis window by as much as 9 days, which is significant because antibodies could not be detectable until 6-7 days after symptoms begin [1].

How long the virus stays in the body depends on a number of factors, including the patient's immune response, the severity of their symptoms, and the duration of their sickness. It takes 21–35 days after symptoms appear or 3–5 days after a patient stops experiencing symptoms for viral shedding to be detected. A molecular test will not pick up on an earlier infection, no matter how long ago it went away, because the viral load becomes undetectable as the disease progresses. While SARS-CoV-2 molecular point-of-care tests are expanding in availability and offering quicker results, the only tests that are now accessible necessitate a laboratory analyzer platform, which is sometimes unavailable or has long wait periods. Numerous labs still have a backlog of samples waiting to be molecularly tested, and public health authorities have instituted stringent criteria that stipulate very severe symptoms as a prerequisite for undergoing a molecular test [1].

As a result, antibody testing remains an essential diagnostic technique for COVID-19. When deciding whether or not to return to community activities or the job, people can benefit greatly from learning their functional immunity to the current epidemic through antibody testing. Antibodies that bind as well as those that neutralize can be sought for. The former have been shown to reduce SARS-CoV-2 viral infection of cells in a laboratory setting by binding to a specific region of the pathogen. The second type of antibody, known as a binding antibody or a non-neutralizing antibody, attaches itself particularly to infectious microbes but does nothing to stop them from spreading. In contrast to neutralizing antibodies, which prevent pathogens from entering cells, binding antibodies indicate that a pathogen is present in the body. While IgG indicates either a present or past illness, IgM indicates an early stage infection [20].

IgG has a half-life of about three months, but IgA and IgM only have a half-life of around two months. Detectable neutralizing antibody titers were present for a shorter duration in some people, whereas in others they remained >1000 at >60 Days Post Symptom Onset (DPSO) [21].

Complement components:

Serum complement C3 and C4 concentration measurements aid in the detection and tracking of immune complex disorders and blood-associated infections. There is a lot of interest in evaluating the complement system during SARS-CoV-2 due to the possible harm that could come from an uncontrolled activation of the system to various organs and tissues' structural and functional integrity. Research showing that corticosteroid medication helps COVID-19 patients provide credence to the idea that the virus isn't the direct cause of organ and tissue damage, but rather an overreaction by the host immune system. In turn, this causes intravascular coagulation and cell death, as shown by complement system activation and the subsequent release of proinflammatory cytokines [22].

Excessive complement activation is deleterious in COVID-19 patients, according to studies, which have revealed that complement components accumulate in the kidneys and lungs and that these organs also show signs of tissue injury. For the second, it's probable that complement C5 convertases are formed with the help of C3b, which is an end product of the C3 convertases. In turn, these break down C5 into C5a, an anaphylatoxin that raises pro-inflammatory pathway activity, and C5b, which initiates the subsequent steps of complement activation, such as the creation of the membrane-attack complex and, through the formation of C5b-9, the induction of cell injury that includes the endothelium [23].

Rheumatoid Factor (RF):

The coronavirus SARS-CoV-2 causes the respiratory infection COVID-19, which is comparable to viral pneumonia. The anti-COVID-19 treatment regimen is heavily reliant on chloroquine and hydroxychloroquine, which are also standard treatments for malaria and autoimmune disorders including rheumatoid arthritis (RA). A large body of research suggests that SARS-CoV-2 infection may be associated with RA. The iatrogenic consequences of RA-related pharmacological therapy put people at a higher risk of infection than the general population. This is especially true for those who are predisposed to RA. Therefore, complicated disorders like RA may be more vulnerable to the COVID-19 pandemic's potential to cause health emergencies [24].

New research shows that SARS-CoV-2 causes a distinct form of alveolar illness, setting it apart from other cases of acute respiratory syndrome. The main factors that lead to severe lung illness in COVID-19 individuals are immune hyperactivation and cytokine involvement in alveolar tissues [25].

Chronic autoimmune illness known as rheumatoid arthritis (RA) causes inflammation of the synovium and overactivity of T cells. In RA, synovial inflammation develops due to many pro-inflammatory cytokines. Patients with COVID-19 exhibit immunological activation and cytokine patterns that are similar to those in RA. Fascinatingly, cytokine suppression and other common treatment approaches have shown promise against both COVID-19 and RA. Therefore, it is quite probable that COVID-19 and RA will engage in pathological crosstalk [25].

Antiphospholipid Antibodies:

The autoimmune disease known as antiphospholipid syndrome (APS) is marked by moderate thrombocytopenia, recurrent foetal losses, and numerous bouts of venous and arterial thromboses or aPL Abs against β2GP1 [26].

In extremely rare cases, APS can lead to catastrophic APS or fulminant multiorgan failure (CAPS). Multiorgan failure due to pervasive small-vessel occlusions is a hallmark of chronic obstructive pulmonary syndrome (CAPS). In a global investigation of CAPS-registry patients' lupus anticoagulant, the most often implicated anti-

phospholipid antibodies (aPL Abs) were IgG anticardiolipin and IgG anti β2GP [27].

Despite only affecting 1% of APS patients, CAPS can be deadly if not treated quickly. Despite the fact that it is clinically significant and has an estimated death rate of 40%, the pathogenesis is still not fully understood. The complete clinical presentation of APS requires complement activation. Serum from APS activates the complement system in vitro, and mutations in the complement gene are common in CAPS patients. The leading cause of CAPS, according to a descriptive study of 500 individuals, was infection [28].

The onset of CAPS can be precipitated by infections, such as COVID-19. Although no proof has been produced, it is plausible that SARS-CoV2 can induce CAPS. Critically sick patients with COVID-19 were found to have aPL Abs at high rates. It is known that critically ill patients with COVID-19 exhibit a severe hypercoagulable condition. Thrombophilia caused by COVID is still not fully understood, however it is believed to contain multiple enteropathogenic factors. Although hypoxia and extended immobilization play a significant role as predisposing variables, the main triggers appear to be the diffuse inflammatory state and the ensuing "cytokine storm" [29].

One of the proposed mechanisms is endothelial dysfunction, which is defined by elevated von Willebrand factor levels; another is systemic inflammation; a procoagulant state; and tissue factor-pathway activation. The stimulation of APLAb synthesis in severe COVID-19 is another potential unrecognized mechanism. [30].

Secondary APS and CAPS may potentially be caused by molecular mimicry that results in the development of anti-β2GP1 Ab. Increased aPL Abs were seen in a small case series of COVID-19 patients, and CAPS is linked to persistently high levels of APL Abs [30].

Studies with COVID-19 patients found that APAs may be a factor in thrombosis based on clinical and laboratory evidence. Notably, among the rare causes linked to both arterial and venous thromboembolic events, as observed in COVID-19 infection, is antiphospholipid syndrome, whether primary or secondary. The importance of investigating the function of APAs in COVID-19 patients has been bolstered by these first results [31].

Patients with COVID-19 have been reported to experience thromboembolic events, such as venous and arterial thromboembolism. There is a substantial death rate linked with these thromboembolic events in COVID-19 [32].

Although there have been reports of high levels of APL Abs in COVID-19 patients, researchers are still trying to determine whether being positive for APA increases the risk of thrombotic events or death from the virus. To date, there has been no discovery of a link between APA positive and in-hospital mortality. While APAs are known to be present in COVID-19 patients, nothing is known about how long they last or what effect they have on diagnosis and treatment. In addition to hepatitis C, cytomegalovirus (CMV), Epstein-Barr virus (EBV), adenovirus, parvovirus, and human immunodeficiency virus (HIV), other illnesses have also been linked to LA- and aCL-positive titers [33].

CONCLUSION

In the absence of evidence from a real-time polymerase chain reaction or antibodies, a thorough familiarity with the laboratory findings and hematological abnormalities linked to SARS-CoV-2 infection might aid in raising suspicion of illness. Rapid screening, diagnosis, prognosis, and treatment of COVID-19 patients could be aided by blood counts in addition to morphological alterations in peripheral blood. Both the diagnosis and the prognosis depend on the thrombocytopenia count. Infection with COVID-19 is indicated by low leukocyte and neutrophil counts, but the opposite is true for progressive infection: increased levels.

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