General Article

HEMATOLOGICAL AND BIOCHEMICAL CHANGES DUE TO EFFECT OF COVID-19 NON-SURVIVORS

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Introduction

Coronavirus disease 2019 was first reported in Wuhan city, Hubei, China, in the last week of December 2019. (1) Patients with coronavirus disease demonstrated a series of clinical symptoms, including raised body temperature, cough, headache, nausea, vomiting, anorexia, diarrhea, dyspnea, multiple organ dysfunctions.(3) A large proportion of infected patients reported mild symptoms of the disease and recover.(4) Some patients progressively develop serious complications, including sepsis, acute respiratory failure, metabolic acidosis, heart failure, kidney injury, hypoxic encephalopathy, and eventually die of the illness.3 A recent report reported a few new symptoms, including anosmia and ageusia.(5) Earlier existing literature on laboratory-confirmed coronavirus cases reported changes in the patients' biochemical parameters, including lymphocyte count, neutrophil count, and D-dimer status.(4,9)Another study reported changes in inflammatory markers in patients with COVID-19, including C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and Interleukin-6.3 Likewise, another work reported lymphocytopenia, high blood sugar, gamma-glutamyl transferase (GGT), high lactate dehydrogenase (LDH) in more COVID-19 patients.(10) Further, laboratory findings of 77 COVID-19 deaths and 852 COVID-19 patients also demonstrated an increase in urea, cardiac troponin, creatine kinase, D-dimer, C-reactive protein (CRP), lactate dehydrogenase (LDH), IL-6, and lower level of lactic acid levels and lymphocytes.(11,12) Analysis of 143 cases of COVID-19 revealed a higher level of C-reactive protein, D-dimer, lactate dehydrogenase, serum amyloid, and lower level of albumin are directly associated with developing a critical illness.(9)

Hematological analysis

Blood cell analysis showed that neutrophil counts were significantly higher in the death group than survivors . Besides, the lymphocyte count remains significantly higher in the survivors' cohort. However, there is no significant difference in white blood cells and platelet count between the survivors and death cohort. In terms of hemoglobin estimation, the survivors' cohort significantly reported a higher hemoglobin level than the death cohort. Besides, there was no significant difference in the D-dimer level between the groups . Further, the hematological profile is compared based on x-ray findings in patients with COVID-19. Findings showed that white blood cells, neutrophils count, and platelet count was significantly higher among patients with abnormal X-ray findings than their counterparts (all $P < 0.05$). However, the lymphocyte count was significantly higher in the cohort with normal x-ray findings ($P < 0.05$). Besides, there was no significant difference in hemoglobin in both the cohorts ($P < 0.05$). The duration of partial thromboplastin and activated thromboplastin time was significantly higher among the cohort reported abnormal

2. Blood biochemical and coagulation profile analysis

Analysis of the biochemical profile showed that the death cohort significantly has higher serum

potassium than the survivors' group. However, there was no significant difference observed in serum sodium and chloride level in both the groups . Besides, the level of aspartate transaminase (AST)) and alanine transaminase (ALT) reported to be significantly higher among the death cohort indicates more severe liver injury in the group. However, there was no significant changes observed in the status of direct bilirubin, indirect bilirubin, and total bilirubin between the two groups. C-reactive protein and procalcitonin level reported significantly higher among the death cohort in contrast to survivors. However, there was no significant difference observed for CPK-MB level in both groups. Further, the level of prothrombin activated prothrombin time and lactate dehydrogdehydrogenase (LDH) reported significantly higher in the death cohort. In contrast, the level of partial oxygen pressure (PaO2) and oxygen saturation (SpO2) reported significantly lower in the death cohort than in survivors hospitalized in ICU.

Discussion

With the continuous spread of COVID-19 cause the effect on different biochemical and hematological profiles in patients who survived or died due to COVID-19. This study showed that the median age of the patients who died with COVID-19 was higher than the survived cohort. These findings are in agreement with the earlier research on patients with COVID-19.(2,19)The older population have more number of comorbidities, limited organ function, reduced lung capacity, impaired immune system, biological aging, and more severe complications, these are the common reason pointed in earlier research on elderly with COVID-19(20,21). Comparison of hematological findings showed that the death cohort had higher neutrophils count, white blood cells, higher prothrombin time, and activated partial thromboplastin time. Besides, the death cohort had lower lymphocyte count, platelets, and lower hemoglobin than survivors' cases. This conclusion mostly matches earlier studies conducted on patients with COVID-19.(2,18,22) There were also significant differences among alanine transaminase, aspartate transaminase, C-reactive protein, procalcitonin, and lactate dehydrogenase between survivors and death cohorts, which are similar to earlier research on COVID-19.(2,9,24) Earlier studies said that level of procalcitonin remains normal in patients with viral infections.(25) In our findings, the procalcitonin level was significantly higher in the death cohort than that of the survived group indicate the possibility of multiple infections at a time. Besides, we found a low level of serum potassium in COVID-19 cases. These findings agree with a study that reported a negative correlation of potassium with the disease severity in COVID-19 cases.9 This is because a higher level of ACE 2 degradation impairs renal control on potassium and makes it challenging for the clinicians to manage hypokalaemia in a patient with SARS-COV-2.

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