Complications of Renal Failure and Toxoplasma gondii Infection on Some Biochemical Parameters of a Patient with COVID-19

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Abstract. This study was designed for infection with *T. gondii* by using specific IgG and IgM by VIDAS technique to indicate *T. gondii.* The experiment was distributed into three groups: (A) (N=30) kidney failure patients group, (B) (N=30) patients with kidney failure patients and COV-19, (C) (N=30) patients with covid- 19 + kidney failure and *T.gondii* infected. The criteria were studied Blood Urea (B.U.) mg\dl, Serum Creatinine (S.Cr.) mg\dl, Albumin serum (ALB) mg\dl, Total serum protein (T.S.P) and phosphate test (PO4) mg\dl. The examination of the parameters mentioned above was performed using a spectrophotometer device by wavelength 490-540 nm. It was seen that the amounts of creatinine, PO4, and blood urea were significantly higher ($P < 0.05$) in COVID-19-infected patients with renal failure than in the control group of patients with renal failure. However, it was noted that patients with COVID-19 and kidney failure, as well as T. gondii infection, demonstrated a return to normal blood urea levels. Creatinine and phosphorous oxyhydroxide levels were not different between those with COVID-19 + renal failure and those with T.gondii infection ($P > 0.05$).

1 Introduction

Toxoplasma gondii is a significant parasite that can only be detected within infected cells and affects one-third of the global population. It eventually makes its home in people's heads. Intracellular parasite belonging to the family Felidae that uses humans as intermediate hosts [1]. In most situations, patients have no symptoms related to feeling [2]. The central nervous system (CNS) is the initial target of a *T.gondii* infection in mammals, birds, and reptiles; the skeletal muscles and reproductive system are secondary targets [3]. Toxoplasmosis, which is spread by this parasite, may be fatal for organ transplant recipients. Foetuses of many other species, both wild and domestic, are also susceptible to contracting congenital toxoplasmosis as a result of this parasite [4]. Pathogenicity of *T.gondii* strains is also host-dependent, with the first type being more commonly isolated from people and the second type being found

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only in the most severe cases of infection [5]. Over the last twenty years, two significant outbreaks have occurred as a result of coronaviruses, namely Severe Acute Respiratory Syndrome (SARS) and Middle East Respiratory Syndrome (MERS). Additionally, a new coronavirus infection, known as SARS-CoV-2, emerged in Wuhan, China and has since spread globally. According to [7], the issue of [public health concern] continues to persist on a global scale, as seen in December 2019. Despite COVID-19 predominantly impacting the respiratory system, research indicates that renal involvement is often seen, including a spectrum from mild proteinuria to severe acute kidney injury (AKI) [8]. Chronic kidney disease (CKD) has become a global pandemic, with an estimated frequency of 14% in the US and 5-15% worldwide [9]. The primary clinical presentations of COVID-19 include symptoms such as elevated body temperature, respiratory distress, weariness, and a persistent cough [10]. A considerable majority of patients had organ function dysregulation, most often respiratory and cardiac [11]. There is evidence suggesting that kidney function may be negatively affected after infection with MERS-CoV and SARS-CoV [12]. Additionally, the coronavirus infection has been associated with impairments in several organs such as the lungs, liver, intestines, and central nervous system [13]. Virus-mediated injury, cytokine storm, Angiotensin II pathway activation, complement disruption, hypercoagulation, and microangiopathy are some of the challenging processes that occur in COVID-19 patients [14]. Multiple studies have shown that there is an increasing clinical indication of Covid-19 transmission in individuals with renal impairment. Proteinuria, in conjunction with hematuria, elevated blood urea nitrogen levels, and heightened serum creatinine levels, is the prevailing clinical manifestation, impacting over 50% of those diagnosed with COVID-19 [15].

The presence of SARS-CoV-2 has been detected in urine and postmortem renal tissue samples of individuals affected by the virus, suggesting that the kidney serves as a specific site of infection for these viral particles [16]. Pathologically, afflicted renal tissues have been shown to exhibit inflammation, edoema, and reduced density [17]. However, current information indicates that Acute Kidney Injury (AKI) is more prevalent among those who are severely unwell. Individuals diagnosed with COVID-19 [18]. Prior studies have shown a varying range of acute kidney injury (AKI) incidence in individuals diagnosed with COVID-19, spanning from 0% to 36.6% [19]. Phosphate levels seem to be protective against lung injury in people with COVID-19 who do not have chronic renal disease [20] Hypophosphatemia has been linked to severe lung damage, but increasing blood phosphate levels have been linked to improved computerized tomography (CT) scans of lung results. COVID-19 severity may be associated with low phosphate levels, according to [21],

In a similar vein, a notable percentage of patients in the COVID-19 study had acute kidney damage (AKI) during the course of the experiment. Acute kidney injury (AKI) was seen with notable frequency among individuals with severe symptoms, manifesting in 29% of such cases, in contrast to its occurrence in a mere 1.2% of non-severe instances [22]. Several studies have shown a link between the new COVID-19 and renal problems [23]. Although the underlying mechanisms of kidney destruction in COVID-19 remain unknown, it has been established that co-morbid conditions may play a role in the disease's progression. Renal complications may manifest at any point throughout the disease and are often referred to as delayed occurrences. In relation to this matter, there was a significantly higher incidence of kidney damage seen in those with renal comorbidities compared to those without (54.5% vs 2.0%, $p<0.001$) [24].

2 Materials and Methods

The present study employed a sample size of ninety (90) male participants, ranging in age from 26 to 68 years. Data collection took place between September and February 2021 at the

kidney centre affiliated with the education leader hospital in AL-Najaf AL-Ashraf Governorate, Iraq. The participants were intentionally infected with T. gondii, and the presence of T. gondii was determined using specific IgG and IgM antibodies through the VIDAS technique. The study design was distributed into three groups: (A) (n=30) kidney failure patients group (control), (B) (n=30) kidney failure patients and COV-19, and (C) (n=30) COV-19 + kidney failure infected+*T.gondii*. The variables of the current study included sugar blood (RSB) mg\dl, Blood Urea (B.U.) mg\dl, Serum Creatinine (S.Cr.) mg\dl, Albumin serum (ALB) mg\dl randomly. Total serum protein (TSP) and phosphate test (PO4) mg/dl. Blood samples from renal failure patients before dialysis were drawn intravenously and placed in test tubes. All patients showed positive coronavirus PCR results by nasopharyngeal swab within 48 hours [6]. Blood was taken and allowed to clot in non-anticoagulant tubes. Centrifugation at 3000 rpm for 10 minutes separated the Serum from the clot. Serum was collected in Epindroff tubes and maintained at -20 °C for the study of biochemical markers such as (randomly sugar (RSB) mgdl, Blood Urea (B.U.) mgdl, Serum Creatinine (S.Cr.) mgdl, Albumin serum (ALB) mgdl. Total serum protein (T.S.P.) and phosphate test (PO4) mgdl) were measured using a spectrophotometer at a wavelength of 490 - 540 nm. The findings were acquired by following the process for each analysis [25].

Statistical analysis: The data were provided in the form of mean ± SEM and were subjected to statistical analysis using the analysis of variance (ANOVA) test. Differences between means were declared significant at $P<0.05$ using the Least Significant Difference Test Analysis of Variance and all other tests were performed using the SPSS (2006) statistical program [2].

3 Result

The results of this investigation revealed a statistically significant increase ($P < 0.05$) in creatinine and blood urea levels in persons with renal failure who were afflicted with COVID-19, as compared to those with renal failure who did not exhibit the infection (the control group). However, it was observed that the blood urea level returned to baseline during the recovery period one month after COVID-19 infection when comparing it to the group of individuals with renal failure who were also infected with COVID-19. While non-significant differences ($P > 0.05$) between the creatinine value COV- 19 + kidney failure infected+*T.gondii* group. So, observe no correlation between total serum protein and albumin serum level between kidney failure patients in the COVID-19 recovery group. There were no discernible distinctions seen between patients with renal failure caused by COVID-19 and those infected with both COVID-19 and kidney failure in conjunction with T. gondii. Nevertheless, a statistically significant increase ($P < 0.05$) in phosphate (PO4) levels was observed in COVID-19 patients when compared to the control group at the same time point.

Table 1. Blood parameters in studied groups

Parameters	kidney failure infected only (control)- n=30 (mean ± SE)	covid- 19 + kidney failure infected -n=30 (mean ± SE)	covid- 19 + kidney failure infected+*T.gondii* -n=30 (mean ± SE)
B.U	182.333 ± 7.995 a	194.2333 ±7.98543 b	175.2667 ± 6.99128 ab
S.CR	7 .1100 ±0.23898 a	9 .1567 ± 0.18844 ab	8.9933 ±0.25460 ab
ALB	3.1233±0.1408	2.7300±0.05615 a	3.0167±0.07239
T.S.P	6.7700± 0.16253	6.8667±0.07912	6.9233±0.08546
PO4	5.6033 ±0.19097 a	6.6667± 0.22114 ab	6.1667 ±0.19589 a

The results are present as mean± SE
a: significant at ($p<0.05$) in comparison with the Renal failure group (control) .
b: significant at ($p<0.05$) in comparison with covid- 19 + kidney failure infected +*T.gondii.*

4 Discussion

T.gondii infects and replicates inside immune cells that it infiltrates. Because immune cells may transport the parasite to the brain, muscles, and other tissues able to maintain a long-term infection, the parasite has the potential to spread throughout the body [4]. Chronic infection is characterized by parasites switching to a bradyzoite transcriptional program and the elimination of the majority of tachyzoites. The parasite must produce a saccharide-rich parasite cyst wall in order to survive passage through the digestive system of the subsequent host [35]. Due to damage to the tissues and organs like the kidney, a change may occur in the values of some physiological parameters, such as enzymes and some Blood elements, which are present in most cells and tissues of the body, especially the liver, kidney and muscles [34]. Studies have indicated that the levels of these elements change when infected with *T.gondii* infection.

The Covid -19 is one of the most widespread viruses in the world, and it has been observed through studies that people with chronic diseases such as kidney disease and the elderly are more susceptible to severe disease [6]. A notable finding emerged indicating that a significant percentage of individuals admitted to medical facilities had Acute Kidney Injury subsequent to contracting the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection [27]. The current study highlights some biochemical variables such as blood urea (B.U.), blood creatinine, serum albumin, total serum protein and phosphate (PO4), when infected with the Covid 19 and COVID-19 + kidney failure infected+*T.gondii*. The findings indicate a statistically significant elevation ($p < 0.05$) in levels of creatinine and blood urea among individuals with renal failure in the COVID-19 group, as compared to the control group consisting of individuals with kidney failure. However, the return of the level of urea in the blood was observed COV- 19 + kidney failure infected+*T.gondii* group. Our findings matched with [35] who found that individuals with COVID-19 had quick increases in urea and serum creatinine levels, as well as significant elevations in uric acid and phosphorus and rapid declines in serum albumin levels [35]. Our findings agreed with prior findings about the decrease in albumin levels in patients with COVID-19 [28]. However, we agreed with another study that observed a clear hypoalbuminemia COV- 19 + kidney failure infected + *T.gondii* [29]. Serum total protein differences between renal failure with covid 19 and the recovery group and the control were not found. Reverse the results that COVID-19 is connected with renal failure and lowers total protein levels [30].

Biochemical markers including albumin, urea, creatinine, and po4 were affected by the Covid-infection, which may account for the findings. Albumin, a vital serum protein generated by liver cells, plays a crucial function in human health and is one of several proteins the liver is responsible for manufacturing. Therefore, a low albumin level is a serious clinical indicator of COVID-19 infection. The production of key acute phase cytokines from blood vessels during cytokine storm has also been linked to liver damage, which is more common in individuals with severe types of COVID-19 infection [31]. Coronavirus viral infection may produce anorexia, which in turn may reduce albumin synthesis [32]. The influences of COVID-19 therapies on kidney functioning and other organs, including the liver, may instead originate through increased vascular permeability, enabling albumin and total serum protein to move into the extravascular space [33]. It also agreed with another research that blood creatinine rose in Covid patients with renal failure and that the rise continued even two weeks after the patients were discharged from the hospital. Furthermore, they observed that among COVID-19 patients, male gender, older age, and high blood pressure were three statistically independent risk variables for renal impairment [22].

As explained study, there were effect of these medications to treat cov2 on kidney functions must be taken with great care it is because of increased renal damage secondary to kidney failure illness [10]. Alternatively, it may be the weak immunity of patients with kidney

failure, giving of chance of the virus an opportunity to enter the kidney tissue and other organs and increase the estimated glomerular filtration rate, in addition to damages in renal tubules, which led to its destruction. According to one study, some severe instances of COVID-19 exhibit symptoms of renal impairment, even in patients who have no previous underlying kidney illness [32]. Our opinion is consistent with the World Health Organization's observed increase in complications after recovery from covid 19 infection period week to month, as body pain and high blood sugar, loss of taste, lack of immune system response, muscle weakness and pancreatitis [9].

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