

Comparison of levels BNP and NT-proBNP between heart failure patients and heart failure with COVID-19

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Abstract

Comprehensive studies have been undertaken on biomarkers linked to coronavirus illness 2019 (COVID-19) in both healthy persons and those with diverse disorders, including cardiovascular diseases. The aim of the current study was to investigate B-Type Natriuretic Peptide (BNP), N-Terminal Pro-B-Type Natriuretic Peptide (NT-proBNP), Cardiac troponin, and certain cytokines, including Interleukin-10 (IL-10), Interleukin-12 (IL-12), and Tumor Necrosis Factors (TNF- α). Study was conducted from January 2022 to August 2022, with participants aged 18 to 68 years. The total number of subjects was 90 individuals: 35 patients with heart failure (G1), 33 of whom had heart failure with COVID-19 (G2), and 22 in the control group (G3). The specimens were obtained from General Kirkuk Hospital and Azadi Teaching Hospital. BNP (brain natriuretic peptide), NT-proBNP (N-terminal pro-BNP), IL-12, IL-10, and TNF- α . The present study showed increase NT-proBNP increased in both G1 and G2 as compared with G3 that were (216.39 $\pm$ 24.43, 213.35 $\pm$ 22.0, 169.50 $\pm$ 23.30) respectively, at P-value <0.001. While BNP highly increased in G2, followed in G2 groups and decreased in control that were (172.25 $\pm$ 21.46, 140.66 $\pm$ 21.24, 85.84 $\pm$ 26.89) respectively, at P-value <0.001. IL-12, TNF-a, IL-10 in G2 were (451.19 $\pm$ 13.5, 144.1 $\pm$ 71.2, 192.0 $\pm$ 25.5) as compared with control that were (350.07 $\pm$ 22.03, 104.1 $\pm$ 144.1 $\pm$ 119.22 $\pm$ 29.6) at p-value<0.05, This study found that covid-19 patients have higher NT-proBNP, BNP and interleukin-10,-12, and TNF-a levels, which indicate illness progression.

Introduction

Heart failure is a diverse illness, making case identification and patient classification in epidemiological studies difficult[1]. COVID-19 was first identified in late 2019 and has expanded worldwide. It is a dangerous respiratory infection. The World Health Organization reports that there have been over 6.8 million deaths caused by the disease, and that there have been over 760 million confirmed cases[2, 3]. The detection of abnormalities is the most important application of biomarkers of people who may be about to deteriorate. The increased morbidity and mortality rates associated with COVID-19 infections make this all the more important. Since the beginning of the COVID-19 outbreak, there has been widespread recognition of the need to use cardiac prognostic signs to identify persons who are at risk of severe infection. Myocardial damage detected in postmortem examinations of cardiac tissues

demonstrates a strong correlation between cardiac biomarkers and the progression of COVID-19 infection [4-6].

N-Terminal Pro-B-Type Natriuretic Peptide (NT-proBNP) and B-Type Natriuretic Peptide (BNP) are biomarkers that can be used to diagnose cardiac damage. These markers are shown to be considerably elevated in patients admitted to the Intensive Care Unit with severe COVID-19 infection[7, 8]. Heart failure, arrhythmia, and myocardial damage are all symptoms that might indicate a more serious cardiac condition. Atrial natriuretic peptide (ANP) is the name given to NPs released by the atrium, while BNP is the name given to NPs secreted by the ventricles[9, 10]. These NPs coordinate essential processes in the cardiovascular and renal systems, including the regulation of electrolyte balance and the maintenance of normal blood pressure [11]. BNP and ANP are secreted in response to stress in ventricular and atrial myocytes, respectively. The endogenous creation of nanoparticles in the brain, kidney, and heart is crucial for regulating the systems that govern cardiovascular functioning [12].

Cardiac troponin concentration is the most favored indicator of damage to the heart muscle. High levels of cardiac troponin are strongly linked to a negative outlook in patients with acute coronary syndromes. They are also used to identify patients who are likely to benefit from an early invasive management approach[13].As endogenous ligands, neuropeptides bind to specific receptors on the brain. Because the COVID-19 infection and the subsequent increase in pulmonary pressure mostly affect the lungs, this condition puts stress on the right ventricle. Atrial distension and the accompanying release of ANP can be caused by elevated pulmonary pressure. Interleukins (IL), particularly IL-10, IL-12,; TNF- α ; and markers from a clinical routine exam evaluations, including D- dimer and C-reactive protein, are recognized biomarkers indicative of an augmented inflammatory response during SARS-CoV-2 infection [14].

Patients infected with COVID-19 exhibit a wide range of immunological profiles, each with varying degrees of variation. The presence of increased amounts of pro-inflammatory cytokines in the bloodstream was found to be connected with a more rapid course of the illness. It has been demonstrated through research that the "cytokine storm" is directly connected to pulmonary damage, failure of multiple organs, and negative outcomes in persons who are infected with COVID-19 [15]. One of the primary functions of the multifunctional cytokine known as interleukin-10 (IL-10) is to reduce the severity of the inflammatory response. Furthermore, increased levels of IL-10 have the potential to act as a stimulant for the activation of the immune system, which in turn can facilitate the creation of additional mediators that are involved in the cytokine storm [16]. Individuals who have COVID-19 may have a more unfavorable prognosis if they have a higher level of cytokine production, which may include IL-12 and IL-15 [17]. When combined with other cytokines, TNF- α is a significant factor in the regulation of inflammatory processes to a significant degree. Patients with COVID-19, particularly those with severe disease, have been observed to have higher levels of TNF- α in their own serum [18]. Within the scope of this study, the objective was to evaluate the possible correlation between the levels of BNP, NT-proBNP, and cytokines, namely IL-10, IL-12, and TNF- α , in conjunction with renal function tests, specifically urea and creatinine, in individuals who have been diagnosed with COVID-19.

The aim of the current study was to investigate B-Type Natriuretic Peptide (BNP), N-Terminal Pro-B-Type Natriuretic Peptide (NT-proBNP), Cardiac troponin, and certain cytokines, including Interleukin-10 (IL-10), Interleukin-12 (IL-12), and Tumor Necrosis Factors (TNF- α).

Materials and Method

Subject

Study was conducted from January 2022 to August 2022, with participants aged 18 to 68 years. The total number of subjects was 90. The overall number of subjects was 90 individuals: 35 patients with heart failure (G1), 33 of whom had heart failure with COVID-19 (G2), and 22 in the control group (G3). The specimens were obtained from General Kirkuk Hospital and Azadi Teaching Hospital. BNP, NT-proBNP, IL-12, IL-10, and TNF- α .

Sample Collection

All individuals, including controls, had their venous blood samples taken. The blood was drawn into a tube using a standard technique called antecubital venipuncture. The 5 ml of remaining blood was transferred to an EDTA-free tube and left to coagulate in a water bath set at 37°C. Centrifugation was then used to separate the serum from the blood samples; after 10 minutes at 3000 rpm, the serum was separated into two tubes and kept at -20°C until analysis. The serum NT-proBNP, BNP, IL-10, IL-12, TNF- α were assessed after the samples were allowed to thaw at room temperature.

Measurement human BNP, cTn, and NT-proBNP by Sandwich-ELISA A kit

The Micro ELISA strip plate that is included in this kit has already been pre-coated with an antibody that can identify BNP, cTn and NT-proBNP in a unique and selective manner. Each well receives an addition of an antibody that has been conjugated with horseradish peroxidase (HRP). Spectrophotometry is used to conduct the measurement of optical density (OD) at a wavelength of 450 nanometers.

Human IL-12, IL-10, and TNF- α by Sandwich-ELISA test

Through the utilization of the ELISA technique, the quantities of IL-12, IL-10, and TNF- α were determined and quantified. Antibodies that are specific to human IL-12, IL-10, and TNF- α were present on the plate before it was coated. In order to augment the sample, levels of IL-12, IL-10, and TNF- α are obtained. We found that there was a positive association between the amounts of human IL-12, IL-10, and TNF- α and the hue of the substrate solution. The process concludes with the introduction of an acidic stop solution, followed by the quantification of absorbance at a wavelength of 450 nm.

Statistical investigation

To compare the study groups, the independent t-test was employed, and the data was represented by the mean and standard error at a probability threshold of less than 0.05.

Results and Discussion

The present study (table 1) showed increase NT-proBNP, troponin increased in both G1 and G2 as compared with G3 that were, at P-value <0.05. While BNP highly increased in G2, followed in G3 groups and decreased in control that were at P-value <0.001. IL-12, TNF- α , IL-10 in G2 were increased in G2 as compared with control that were at p-value <0.05.

Table 1: Level of NT-proBNP, BNP, Troponin, IL-12, TNF-a, IL-10 between study groups

Parameters	G1	G2	G3	P-value
NT-proBNP ng/ml	216.39±24.43a	213.35±22.0a	169.50±23.30 b	<0.05
BNP pg/ml	140.66 ±21.24 b	172.25± 21.46 a	85.84 ± 26.89 c	0.001
Troponin(ng/ml)	94±22.15a	97.7±12.42a	50.37±13.69b	0.01
IL-12 pg/ml	443.07±19.0a	451.19±13.5a	350.07±22.03b	0.05
TNF-a pg/ml	108.3± 16.3 b	144.1± 71.2 a	104.1± 14.2 b	0.02
IL-10 pg/ml	145±20.7 b	192.0±25.5a	119.22±29.6 b	0.004

Over the course of this analysis, it was discovered that individuals who were diagnosed with HF and heart failure associated with COVID-19 had higher levels of NT Pro-BNP compared to the control group. The results of this investigation are consistent with the findings of [19], which demonstrate that patients with HF who were infected with COVID-19 had a significantly higher level of natriuretic peptides, notably BNP and NT-proBNP, in comparison to individuals who were not infected with the virus. According to the findings of the study that was carried out by [20], elevated levels of NT-proBNP are mostly associated with the existence and severity of cardiac disease, rather than being the result of decreased renal function. In the context of cardiovascular disorders, such as HF and coronary artery disease (CAD), NT-proBNP has been acknowledged as a significant predictive marker for mortality [21, 22].

The present investigation shows that there is an increase in cardiac troponin levels in individuals with cardiovascular disease compared to the control group. A comprehensive retrospective cohort study observed a positive correlation between elevated cTn levels and a range of cardiovascular risk factors, as well as both cardiovascular and noncardiovascular diseases. However, it is worth noting that only patients with atherosclerosis showed a substantial increase in cardiac troponin levels. This suggests that cardiac troponin can be regarded a biomarker for identifying individuals who are at risk of developing atherosclerosis [23]. Troponin, a cardiac enzyme, plays a crucial role in the contraction of both cardiac and skeletal muscles. Elevated concentrations of troponin T in the bloodstream have been linked to damage in the heart muscle, and these levels have been utilized to assess the extent of a heart attack [24]. Patients with COVID-19 may experience severe repercussions resulting from immunological dysfunction linked to heightened cytokine storms[22]1. In the majority of fatal cases, excessive cytokine storm activation was identified as the cause, leading to the development of acute lung damage and subsequently acute respiratory distress syndrome (ARDS)[25]. A strong inflammatory response involving IL-6 and IL-10 is crucial to COVID-19 development. In sepsis and acute organ injury, tissue inflammation produces pleiotropic

cytokines. Lymphocytes, macrophages, fibroblasts, endothelial cells, and epithelial cells produce these cytokines into the bloodstream [26]. The adaptive immune response shows a significant rise in IL-10 following influenza infection. This applies especially to the response [27].

Our biomarker results align with previously documented correlations between the levels of several cytokines (e.g., IL-2, IL-10, and TNF- α) with the severity and mortality of COVID-19 [28]. The relationship between COVID-19 progression to severe stages and IL-10 levels has been documented in numerous papers[29]. IL10 is an immunoregulatory cytokine primarily responsible for attenuating inflammatory responses and modulating the development and proliferation of immune cells[30]. The information regarding the function of IL-12 in COVID-19 is relatively scarce. Our investigation found a link between high IL-12 and severe COVID-19[31]. A pathogen-associated molecular response induces IL-12 production in cells after a virus enters [32]. IL-12, a heterodimeric cytokine with p35 and p40 subunits, enhances innate and adaptive immune responses. In COVID-19 patients, concentrations of IL-10 and TNF- α were identified as unique markers of T-cell depletion, as documented in the reference[33]. In addition to its many other functions, the cytokine known as IL-10 is principally responsible for reducing the inflammatory response. It has been established that IL-10 is responsible for causing anergy or non-responsiveness in T-cells during anti-tumor responses [34] and viral infections[35]. The elimination of viral infection was accomplished by either genetically ablation of IL-10 or the delivery of an antibody that targets IL-10 or its receptor. Both of these methods were successful in preventing the spread of the virus[18]. Prior to this, it was believed that the high levels of IL-10 that were detected in patients with severe COVID-19 were the product of a negative feedback loop. This was due to the fact that IL-10 possesses anti-inflammatory properties[36]. Considering its pro-inflammatory characteristics. During the onset of viral infection, IL-10 functions as a counter-regulatory agent to pro-inflammatory mediators through a negative feedback mechanism. Nonetheless, as the disease advances, the endogenous level of IL-10 escalates, activating its proinflammatory properties and contributing to the exacerbating cytokine storm[18].

Conclusions

This study suggests a dysregulated immunological response to COVID-19, with elevated levels of TNF- α , IL-12, and IL-10. Cost-effective TNF- α , IL-12, and IL-10 testing can be done upon patient admission to clinics or hospitals with limited resources. This measurement will be essential for identifying patients at elevated risk of advancing to severe disease, facilitating the administration of suitable preventative strategies. Besides its diagnostic significance, the study of this data is expected to produce potential therapeutic techniques aimed at TNF- α , IL-12, IL-10, or a combination thereof. Assessment of BNP, NT-proBNP, Cardiac troponin, concentrations in patients at disease onset and after 2 to 4 days revealed that impaired renal function manifested in COVID-19 patients, representing a primary factor in death within this population.

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مقارنة مستويات BNP و NT-proBNP بين مرضى قصور القلب وفشل القلب المصابين بـ COVID-19

حسين علي محمد البدرى

قسم الكيمياء، كلية العلوم، جامعة ديالى، ديالى، العراق

الخلاصة:

تم إجراء دراسات شاملة على المؤشرات الحيوية المرتبطة بمرض فيروس كورونا 2019 (كوفيد-19) لدى كل من الأشخاص الأصحاء والأشخاص الذين يعانون من اضطرابات متنوعة، بما في ذلك أمراض القلب والأوعية الدموية. كان الهدف من الدراسة الحالية هو دراسة الببتيد الناتريوتريك من النوع B (BNP)، والببتيد N-Terminal Pro-B (NT-proBNP) من النوع (Natriuretic)، وبعض السيتوكينات، بما في ذلك IL-10، IL-12، TNF- α). وأجريت الدراسة في الفترة من يناير 2022 إلى أغسطس 2022، على المشاركين الذين تتراوح أعمارهم بين 18 إلى 68 عامًا. كان العدد الإجمالي للأشخاص 90 فردًا: 35 مريضًا يعانون من قصور القلب (G1)، و33 منهم يعانون من قصور القلب مع كوفيد-19 (G2)، و22 في المجموعة الضابطة (G3). تم الحصول على العينات من مستشفى كركوك العام ومستشفى آزادي التعليمي. BNP (الببتيد الناتريوتريك في الدماغ)، NT-proBNP (N-terminal pro-BNP)، IL-12، IL-10، و TNF- α . أظهرت الدراسة الحالية زيادة NT-proBNP في كل من G1 و G2 مقارنة بـ G3 التي كانت (24.43 $\pm$ 216.39، 22.0 $\pm$ 213.35، 23.30 $\pm$ 169.50) على التوالي، عند قيمة $P < 0.001$. بينما ارتفع BNP بشكل كبير في G2، يليه في المجموعات G2 وانخفض في السيطرة التي بلغت (21.46 $\pm$ 172.25، 21.24 $\pm$ 140.66، 26.89 $\pm$ 85.84) على التوالي، عند قيمة $P < 0.001$. كانت IL-12 و TNF- α و IL-10 في (451.19 $\pm$ 13.5، 144.1 $\pm$ 71.2، 192.0 $\pm$ 25.5) مقارنة بالتحكم الذي كان (22.03 $\pm$ 350.07، 104.1 $\pm$ 144.1، 29.6 $\pm$ 119.22) عند القيمة $p < 0.05$. هذه الدراسة وجدت أن مرضى كوفيد-19 لديهم مستويات أعلى من NT-proBNP و BNP، و IL-10، TNF- α ، مما يشير إلى تطور المرض.

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