Open Access Original Article

Measurement of the serum level of Elabela for the early detection of acute kidney injury in hospitalized Iraqi COVID-19 patients

DOI: https://doi.org/10.32007/jfacmedbagdad.6431928

Maha H. Gadhi* **BSc (Pharma)**
Eman S. Saleh** **PhD**

JFac Med Baghdad 2022; Vol.64, No. 3
Received: June. 2022
Accepted: Aug. 2022
Published: Oct. 2022

Abstract:

Background: Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) is caused coronavirus disease 2019 (COVID-19) affecting people worldwide. The angiotensin converting enzyme2 (ACE2) represents a receptor of SARS-CoV-2 on the infected host cell. Apelin or its receptor agonists suppress the production of angiotensin-converting enzyme (ACE) and angiotensin II (Ang-II) and is characterized by a protective effect against SARS-CoV-2.

Objective: The study aims to assess the serum level of Elabela biomarker as an early detector for Acute Kidney Injury (AKI) in patients with COVID-19.

Cases and Methods: This is a case-control study which included 45 hospitalized adult patients in multiple centers (public hospitals) receiving COVID-19 cases in Baghdad. These cases had a positive real-time or reverse transcription polymerase chain reaction (RT-PCR) of nasal/oropharyngeal swabs. Excluded from the study were those with a negative PCR and comorbidities and 43 apparently healthy adult subjects as controls. The age range of the cases and controls was (20 to 60) years

Result: There are no a statistically significant differences between the two groups in terms of age and gender distribution. Statistically significant differences were found in terms of eGFR, S. Creatinine, D. dimer, NEU×10^3/µL, LYM×10^3/µL and ELA biomarker. Significant negative correlations were found between Elabela with D. dimer and NEU×10^3/µL, and between eGFR with S. creatinine, D. Dimer, and NUT×10^3/µL.

Conclusion: The Elabela biomarker can be used for the early detection of acute kidney injury in COVID-19 patients.

Keywords: COVID-19, ACE2, Acute kidney injury, Elabela.

Introduction:

The term COVID-19 was introduced by the World Health Organization (WHO) in January/2020(1) to name the condition of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection It may be a minor disease or progress to a multi-organ failure causing death(2). The virus is an enveloped RNA virus referred to Betacoronavirus genus(3). The first manifested case of (Covid-19) infection in Iraq was diagnosed on February 24, 2020 in Al-Najaf governorate, in a young man coming from Iran(4). WHO recorded 519 729 804 reported cases, and 6 268 281 reported deaths worldwide update 17 May 2022(5). Symptoms of COVID-19 are fatigue, headache, fever, dyspnea, cough, myalgias, and also can caused diarrhea. In addition to symptoms, the imaging abnormalities could be recognized as COVID-19 infection through chest x-ray and computed tomography (1).

* *Department of Clinical Laboratory Science –College of Pharmacy / University of Baghdad*
Correspondence author's email: maha.hasan1200m@copharm.uobaghdad.edu.iq. emansaadi@copharm.uobaghdad.edu.iq

COVID-19 causes atypical pneumonia and severe respiratory failure which it's leading to may cause Acute Kidney Injury (AKI)(6), with significantly poor outcomes(7). The patients that are more likely to develop AKI are required to be admitted to the intensive care unit (ICU), therefore they required renal replacement therapy. COVID-19-associated AKI is caused by the extensive release of pro-inflammatory mediators, hypoperfusion, renal congestion caused by the increased amount of positive end-expiratory pressure, and viral invasion directly into the epithelial cells of the kidney tissue and podocytes need to be admitted to the intensive care unit (ICU) and are These patients need to be admitted to the intensive care unit (ICU) and are more likely to develop AKI, therefore they require renal replacement therapy. COVID-19-associated AKI is caused by the extensive release of pro-inflammatory mediators, hypoperfusion, renal congestion caused by the increased amount of positive end-expiratory pressure, and viral invasion directly into the epithelial cells of the kidney tissue and podocytes, therefore they require renal replacement therapy.

COVID-19-associated AKI is caused by the extensive release of pro-inflammatory mediators, hypoperfusion, renal congestion caused by the increased amount of positive end-expiratory pressure, and viral invasion directly into the epithelial cells of the kidney tissue and podocytes(8). The enzyme responsible for the conversion of Angiotensin-I (Ang-I) to Angiotensin-II (Ang-II) is the Angiotensin-converting enzyme (ACE) that accumulates in the pneumocytes, endothelial cells (9), heart, and kidney (10). The virus enters the host cell by tissue spike (S) protein of the virus and binds with transmembrane ACE2 of the host cell(9)(11), accompanied by a reduced response to expressing ACE2 and causing a rise in Ang-II production(12) leading to vasoconstriction, activation of the inflammatory response, endothelial damage, and collagen synthesis within fibroblasts, ending in fibrosis in these organs. ACE2 is a counter-regulatory enzyme that decomposes Ang-II forming angiotensin 1–7 causing vasodilation and enhancing Ang-II interfered inflammation (13). ACE2 is presented with an excess amount of renal than pulmonary tissues (14) arranged in the brush border membrane of the proximal tubule and present in small amounts in the podocytes (8). The virus penetrates renal tissues by attacking the podocyte, entering tubular secretions, and attaching within ACE2 at the proximal tubule(15). The apelinergic system act as an essential signaling pathway by the modulation of cardiovascular homeostasis. Its G protein-coupled receptor which shares the same tendency of tissue deposition coupled to apelin peptide jejunum (APJ) is defined as the angiotensin II type 1 receptor (AT1R) and its endogenous ligand (16), Elabela (ELA) is a 32-residue peptide hormone, an endogenous ligand for (APJ) (17). It adjusts fluid homeostasis (18), reduces blood pressure (19), increases the formation of new blood vessels (20), and is well preserved in case of renal insult(21). Studies demonstrate that these peptides may diminish the intensity of Acute Lung Injury (ALI) by reducing the accumulation of fluid in the alveoli, secretion of cytokines, and hypoxemia, thus developing with COVID-19-linked ARDS causing a subsequent insult to the cardiac, renal, and other tissues (22). So that those peptides would inhibit the severe effects of COVID-19, and their favorable effects expand to protecting different body tissues from increased levels of inflammatory cytokines and reducing fatality (23).

Patients and Methods:

The data for this multi-center study was collected from several public hospitals including Al-Kindy teaching hospital, Al-Ataa hospital, and Sheikh Dhari Al-Fayadh hospital, in Baghdad-Iraq from September 2021 to January 2022. This study was approved by the University of Baghdad / College of Pharmacy and the Iraqi Ministry of Health / Rusafa Health Directorate. Hospitalized patients with COVID-19 and with a positive real-time reverse transcription polymerase chain reaction (RT-PCR) of respiratory samples taken from nasal/oropharyngeal swabs (24) were included in this study as the cases. Inclusion criteria were: Adult patients between 20 to 60 years of age with COVID-19 diagnosed clinically with fever and pulmonary symptoms (cough, shortness of breath, chest tightness, and pain), a positive RT-PCR for COVID-19, and radiological findings of consolidation either on chest X-ray or computerized tomography (CT). Exclusion criteria include Negative RT-PCR for COVID-19, pregnant women, and comorbidities (liver, renal, cardiovascular diseases, hypertension, diabetes, and autoimmune diseases). Two groups were included in the study: Group 1: 45 (20 females and 25 males) severe COVID-19 cases with respiratory distress (dyspnea).
Group 2: 43 (20 females and 23 males) were healthy age-matched as control.

Laboratory Analysis

- Five milliliters of blood were drawn from COVID-19 patients. Three ml was placed in a gel tube and left to coagulate for 15 minutes. The samples were then centrifuged at 5,000 rounds per minute (RPM) for 5 minutes and the serum was collected by a micropipette in a plain tube and stored at (- 20º C) to measure Human Elabela (pg/ml) sandwich Enzyme-Linked Immunosorbent Assay (ELISA) kit (25). The remaining serum was used for measuring S. creatinine (mg/dL) by using a multiple wavelength spectrophotometer (26).
- The remaining 2 ml of blood was used as follows: Sodium citrate tube for measurement of D-dimer level in COVID-19 patients. The sample of blood was mixed gently for one minute with an anticoagulant reagent, then centrifuged at 4000 RPM for 6-10 minutes. The collected plasma was used immediately for measurement level D-dimer by using fluorescence immunoassay (27).
- EDTA test tube to prevent coagulation of blood sample. Sysmex /XN-350 analyzer was used to measure the White Blood Cell differential count (28). According to modification of Diet in Renal Disease [MDRD] Study Equation, GFR (mL/min/1.73 m^2) = 175 × (Scr)-1.154 × (Age)-0.203 × (0.742 if female) is used for calculating the estimated glomerular filtration rate (eGFR) (ml/min) (29).

Statistical Analysis:

Descriptive data were presented as numbers and percentages for categorical variables, mean ± standard deviation for normally distributed variables, and median for variables that are not normally distributed. The Chi-square test was used to test associations between variables, the Mann–Whitney U test was used for the group's analysis of non-normally distributed variables and the Student t-test was used to study differences between means of the groups for normally distributed data. Non-parametrical correlation (spearman correlation) was used for assessing the correlation between Elabela biomarker and eGFR with different laboratory parameters including S. Creatinine µmol/L, D. Dimer µg/ml, NEU×103/µL, and LYM×103/µL. Assessing a test's diagnostic performance or accuracy in distinguishing diseased from normal cases was done by using Receiver Operating Characteristic (ROC) curve analysis. All tests were two-tailed, and differences were considered statistically significant at (P-values <0.05).

Table 1: Distribution of the study groups by demographic and lab data

Variables		Median or mean±SD		P - value
		Patients	Controls	
Age (years)		47.0	47	0.923
Gender n. (%)	Male	25 (28.4%)	23 (26.1%)	0.467
	Female	20 (22.7%)	20 (22.7%)	
S. creatinine (µmol/L)		57.8±21.09	42.7±7.67	0.000
eGFR (ml/min/1.73 m2)		136	180	0.000
ELA (pg/ml)		270.9	426.6	0.000
NEU×103/µL		9.8	5.8	0.000
LYM×103/µL		1.2	1.9	0.000
D.D (µg/ml)		1.1	0.1	0.000

Descriptive data were presented as median for non-parametrical variables and mean±SD for parametrical variables.
eGFR: estimated glomerular filtration rate, ELA: elabela, D.D: D. dimer1, NEU×103/µL: neutrophile cell×103/µL, and LYM×103/µL: lymphocyte cell×103/µL

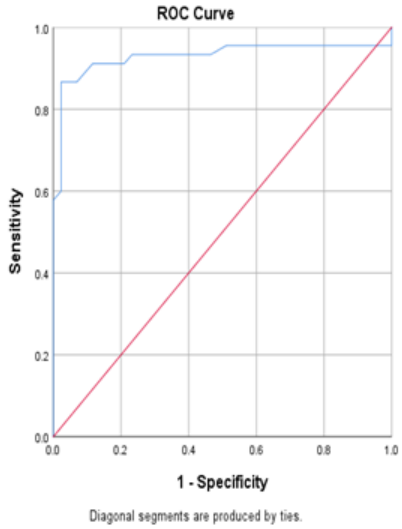

Figure 1: Receiver Operating Characteristic Curve of D-dimer for COVID-19 Patients

Results

This study was conducted on 45 hospitalized COVID-19 patients, 20 (22.7%) females and 25 (28.4%) males, and 43 apparently healthy subjects, 20 (22.7%) females and 23 (26.1%) males. The distribution of the cases and controls by gender did not show any significant associations (p-value =0.467).

Table 2: Correlations between Elabela and eGFR with other variables

Parameter		Elabela		eGFR	
Elabela (pg/mL)	P value	r.	P value	r.	
	-	-	0.386	0.094	
D. Dimer (µg/mL)	P value	r.	P value	r.	
	0.000	- 0.407**	0.000	-0.374**	
S. Creatinine (µmol/L)	P value	r.	P value	r.	
	0.160	- 0.151	0.000	-0.753**	
NEU×103/µL	P value	r.	P value	r.	
	0.000	-0.513**	0.000	-0.385**	
LYM×103/µL	P value	r.	P value	r.	
	0.000	0.380**	0.033	0.227*	

r: correlation coefficient.
** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 2 shows the correlation between Elabela biomarker and eGFR with different laboratory parameters. It shows that significant correlations were found between Elabela and D. dimer (r = - 0.407; p = 0.000), Elabela with NEU×103/µL (r = - 0.514; p = 0.000), between eGFR with S. creatinine (r = - 0.753; p = 0.000), D. Dimer (r = - 0.374; p = 0.000), and NUT×103/µL (r = - 0.385; p = 0.000). Significant positive correlations were found between Elabela and eGFR with LYM×103/µL (r = 0.380; p = 0.000) and (r = 0.227; p = 0.033) respectively.

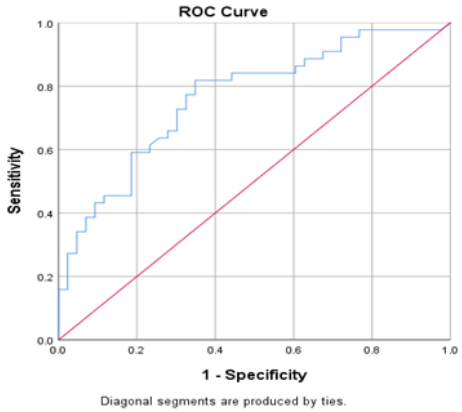

Figure 2: Receiver Operating Characteristic Curve of Elabela for COVID-19 Patients

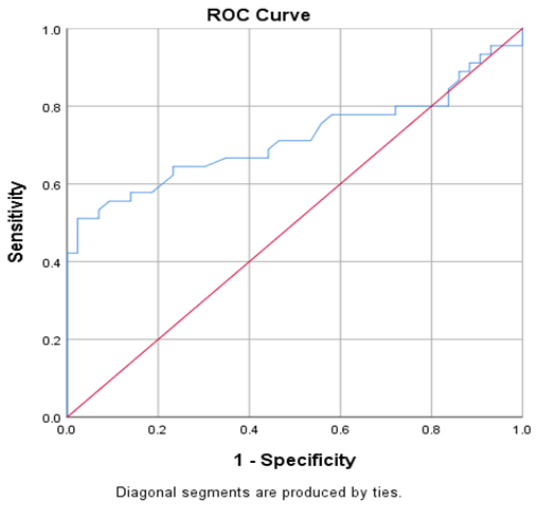

Figure 3: Receiver Operating Characteristic Curve of eGFR for COVID-19 Patients

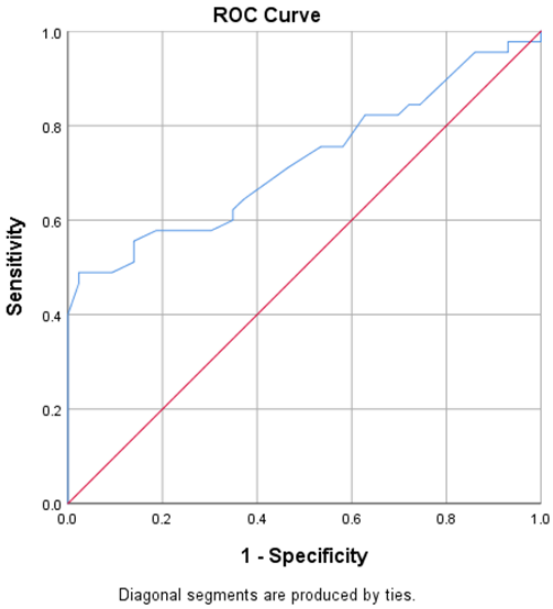

Figure 4: Receiver Operating Characteristic Curve of S. creatinine for COVID-19 Patients

Table 4: ROC Curve Analysis of D-dimer, ELA, eGFR, and S. creatinine in the study groups

Test Variables	Accuracy	Area (AUC)	Significance Asymptomatic	Asymptomatic 95% Confidence Interval	
				Lower Bound	Upper Bound
D.D (µg/ml)	Excellent	0.929	0.000	0.864	0.995
ELA (pg/ml)	Good	0.764	0.000	0.664	0.864
eGFR (mL/min/1.73 m2)	Good	0.717	0.000	0.604	0.829
S. creatinine (µmol/L)	Good	0.725	0.000	0.618	0.832

Table 4 shows the use of receiver operating characteristic (ROC) in measuring the accuracy of D-dimer, ELA, eGFR, and S. creatinine in the studied groups. The area under the curve (AUC) of D.D was 0.929, and the cutoff value of D-dimer is (0.054 µg/ml), as represented in figure (1), area under the curve of ELA is 0.764 and the cutoff value (376.11 pg/ml), as represented in figure (2), area under the curve of eGFR 0.717, and cutoff value (161 ml/min/1.73 m2) as represented in figure (3), and area under the curve of S. creatinine 0.725, and cutoff value (42.88µmol/L) as represented in figure (4) .

Discussion:

This study shows that there were more male COVID-19 cases than females. This agrees with other studies that showed males to be more affected by Covid-19 (72%) than females (28%) (30). A significant difference was found between controls and patients for eGFR and S. creatinine. The patients are admitted to the intensive care unit when their eGFR falls below 60 mL/min/1.73 m2 which is a prognostic parameter for mortality in patients with COVID-19. eGFR decreases with age, even in people without kidney disease, older patients are known to have a higher risk. An elevated level of S. creatinine associated with COVID-19 disease causes admissions of patients to ICU, who should be monitored more carefully for early intervention on admission(31)(32). There was a significant difference in D-dimer between the patients and controls. Elevated level of D-dimer contributed to the severity of the disease(36). The impaired renal function causes the activation of an inflammatory process even if the patient does not develop severe respiratory failure. Therefore, measurement of renal function should be checked. The mild respiratory signs due to dehydration and hypoperfusion, are mostly attributed to AKI following intravenous fluids replacement therapy to control hemodynamics and fluids balance(33). The inflammatory process can cause multiorgan failure in COVID-19 patients(35), the inflammatory process can stimulate the production of platelets and damage the endothelium, causing clotting, coagulation problems, vascular injury, and the elongation of international normalized ratio in patients of AKI(31). Many drugs such as the antivirals which were tested for their effectivity against COVID-19 and the antibiotics which are used for the treatment of secondary bacterial infections in COVID-19 patients have adverse effects on the kidneys and may have a co-adjuvant effect with SARS-CoV-2 itself in increasing the incidence of AKI incidence. Testing kidney functions aid in accomplishing the most appropriate drug therapeutic concentrations and diminishing the risk of adverse drug reaction of most drugs that are prescribed to treat COVID-19(34). This study shows significant differences in lymphocyte count and neutrophil count in Covid-19 patients in comparison with healthy subjects.

Lymphopenia is an important feature of COVID-19 that is more marked in critically ill patients(35). The correlation between eGFR and S. creatinine in the presence of other parameters that include D-dimer, lymphocytes, and neutrophils count is similar to other the findings of other studies, as well as S. creatinine and eGFR in the association of disturbance hematological parameters(37). The serum level of the Elabela biomarker is lowered in COVID-19 patients compared with control group with a significant correlation between Elabela, D-dimer, and lymphocytes and neutrophils count. Apelin and agonists of its receptor increase the activity of ACE-2 leading to inhibition of the effect of the ACE-Ang-II system that produces acute pulmonary insult, coagulation problem, and acute or chronic cardiac insult in COVID-19 patients so that apelin may act as a predictive parameter for the lung and extra-pulmonary insults (38). Interleukin-6 is a pro-inflammatory mediator elevated in COVID-19 patients and plays a role in the severity of COVID-19 infection that complicated AKI.

This study had some limitations including the small number of patients.

Conclusion:

The Elabela biomarker can be used for the early detection of acute kidney injury in COVID-19 patients.

Authors' contributions:

Pharmacist Maha H. Gadhi: conception and design of study, Investigation, Methodology, interpretation and analysis of data, Writing, preparation of original manuscript, Writing Review and Editing.

Prof. Dr. Eman S. Saleh: Supervision, preparation of original manuscript, conception and design of study, Investigation, Methodology, Writing Review and Editing.

Reference

1. Alqubbanchi FB, Al-Hamadani FY. A Pharmacoeconomics Study for Anticoagulants used for Hospitalized COVID-19 Patients in Al-Najaf Al-Ashraf city–Iraq (Conference Paper). Iraqi J Pharm Sci (P-ISSN 1683-3597, E-ISSN 2521-3512). 2021;30(Suppl.):48–59.

2. Allawi JS, Abbas HM, Rasheed JI, Sulaiman TI, Gatea AA, Al-Lami F, et al. The first 40-days experience and clinical outcomes in the management of coronavirus covid-19 crisis. Single center preliminary study. J Fac Med Baghdad. 2019;61(3, 4).

3. Nassi KF. Clinical evaluation of selected Pharmacological Treatments used for Coronavirus (COVID-19) pandemic. J Fac Med. 2020;62(1–2).

4. AL-Shareef AH, Al-Mudhaffer SA, Mohammed S. Epidemiology and clinical characteristics of the domestic and repatriated (Covid-19) Infections in Al Najaf governorate, Iraq. J Fac Med. 2020;62(1–2).

5. WHO. Coronavirus disease 2019 (COVID-19): situation report, 73. 2020;

6. Chen Y-T, Shao S-C, Hsu C-K, Wu I-W, Hung M-J, Chen Y-C. Incidence of acute kidney injury in COVID-19 infection: a systematic review and meta-analysis. Crit Care. 2020;24(1):1–4.

7. Hirsch JS, Ng JH, Ross DW, Sharma P, Shah HH, Barnett RL, et al. Acute kidney injury in patients hospitalized with COVID-19. Kidney Int. 2020;98(1):209–18.

8. Gupta A, Madhavan M V, Sehgal K, Nair N, Mahajan S, Sehrawat TS, et al. Extrapulmonary manifestations of COVID-19. Nat Med. 2020;26(7):1017–32.

9. Mirz AJ, Taha AY. Catheter directed thrombolysis for COVID-19 thrombotic complications in Kurdistan-Iraq: two case reports. J Fac Med. 2021;63(2).

10. South AM, Tomlinson L, Edmonston D, Hiremath S, Sparks MA. Controversies of renin–angiotensin system inhibition during the COVID-19 pandemic. Nat Rev Nephrol. 2020;16(6):305–7.

11. Kuba K, Imai Y, Rao S, Gao H, Guo F, Guan B, et al. A crucial role of angiotensin converting enzyme 2 (ACE2) in SARS coronavirus–induced lung injury. Nat Med. 2005;11(8):875–9.

12. Gu H, Xie Z, Li T, Zhang S, Lai C, Zhu P, et al. Angiotensin-converting enzyme 2 inhibits lung injury induced by respiratory syncytial virus. Sci Rep. 2016;6(1):1–10.

13. Vaduganathan M, Vardeny O, Michel T, McMurray JJ V, Pfeffer MA, Solomon SD. Acute Kidney Injury in COVID-19: Emerging Evidence of a Distinct Pathophysiology. N Engl J Med. 2020;382(17):1653–9.

14. Ye M, Wysocki J, William J, Soler MJ, Cokic I, Batlle D. Glomerular localization and expression of angiotensin-converting enzyme 2 and angiotensin-converting enzyme: implications for albuminuria in diabetes. J Am Soc Nephrol. 2006;17(11):3067–75.

15. Batlle D, Soler MJ, Sparks MA, Hiremath S, South A, Welling P, et al. Acute kidney injury in COVID-19: Emerging evidence of a distinct pathophysiology. Vol. 31, Journal of the American Society of Nephrology. American Society of Nephrology; 2020. p. 1380–3.

16. Tatemoto K, Hosoya M, Habata Y, Fujii R, Kakegawa T, Zou M-X, et al. Isolation and characterization of a novel endogenous peptide ligand for the human APJ receptor. Biochem Biophys Res Commun. 1998;251(2):471–6.

17. Chng SC, Ho L, Tian J, Reversade B. ELABELA: a hormone essential for heart development signals via the apelin receptor. Dev Cell. 2013;27(6):672–80.

18. Deng C, Chen H, Yang N, Feng Y, Hsueh AJW. Apela regulates fluid homeostasis by binding to the APJ receptor to activate Gi signaling. J Biol Chem. 2015;290(30):18261–8.

19. Yang P, Maguire JJ, Kuc RE, Southwood M,

Morrell NW, Davenport A. ELABELA/Toddler (Apela), is an endogenous ligand of the human apelin receptor and is reduced in pulmonary arterial hypertension. Circulation. 2015;132(suppl_3):A13911–A13911.

20. Ha SK. Dietary salt intake and hypertension. Electrolytes Blood Press. 2014;12(1):7–18.

21. Chen H, Wang L, Wang W, Cheng C, Zhang Y, Zhou Y, et al. ELABELA and an ELABELA fragment protect against AKI. J Am Soc Nephrol. 2017;28(9):2694–707.

22. Fan XF, Xue F, Zhang YQ, Xing XP, Liu H, Mao SZ, et al. The Apelin-APJ axis is an endogenous counterinjury mechanism in experimental acute lung injury. Chest. 2015;147(4):969–78.

23. Yang P, Maguire JJ, Davenport AP. Apelin, Elabela/Toddler, and biased agonists as novel therapeutic agents in the cardiovascular system. Trends Pharmacol Sci. 2015;36(9):560–7.

24. Wei PF. Diagnosis and treatment protocol for novel coronavirus pneumonia (trial version 7). Chin Med J (Engl). 2020;133(9):1087–95.

25. Ozsoyler I, Ucak HA, Badak TO, Cakallioglu A, Bayraktar M, Arslan AS. The İmpact Of The Apelinergic System In Coronary Collateral Formation. 2022;

26. Arini A. Evaluasi Proses Maintenance, Controlling Dan Troubleshooting Pada Automated Clinical Chemistry Analyzer Cobas C 311 Di Laboratorium Rumah Sakit Universitas Airlangga. Universitas Airlangga; 2019. A thesis submitted to an Indonesian university. This reference was accessed through Google Scholar

27. Farooqi H, Firdous S, Kazmi S, et al. 2022. Elevated D-Dimer levels are strongly associated with High Mortality Rate in COVID-19 patients. An observational Study: Elevated D-Dimers in COVID-19 Patients. Pakistan Biomed J. 83–9.

28. Khartabil TA, de Frankrijker MM, de Rijke YB, et al. 2021. The Sysmex XN-L (XN-350) hematology analyzer offers a compact solution for laboratories in niche diagnostics. Int J Lab Hematol. 43(1):29–39.

29. Levey AS, Coresh J, Greene T, et al. 2006. Using standardized serum creatinine values in the modification of diet in renal disease study equation for estimating glomerular filtration rate. Ann Intern Med. 145(4):247–54.

30. Hachim IY, Hachim MY, Naeem K Bin, et al. 2020. Male Gender is a risk factor for sever form of COVID-19 illness and worse outcome in the Middle East.

31. Ustundag Y, Engindeniz FT, Huysal K, et al. 2022. Estimated glomerular filtration rate in identifying illness severity in newly admitted patients with COVID-19: A single-center study. Int J Med Biochem. 5(1):44–8.

32. Li Z, Wu M, Yao J, et al. 2020. Caution on kidney dysfunctions of COVID-19 patients.

33. Xia P, Wen Y, Duan Y, et al. 2020.Clinicopathological Features and Outcomes of Acute Kidney Injury in Critically Ill COVID-19 with Prolonged Disease Course: A Retrospective Cohort. J Am Soc Nephrol. Sep 1;31(9):2205–21.

34. Rismanbaf A, Zarei S. 2020. Liver and kidney injuries in COVID-19 and their effects on drug therapy; a letter to editor. Arch Acad Emerg Med. 8(1):e17–e17.

35. Huang C, Wang Y, Li X, et al. 2020. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet. 395(10223):497–506.

36. Alnor A, Sandberg MB, Gils C, et al.2020 . Laboratory tests and outcome for patients with coronavirus disease 2019: a systematic review and meta-analysis. J Appl Lab Med. 5(5):1038–49.

37. Hachim IY, Hachim MY, Naeem K Bin, et al. 2021.Kidney dysfunction among COVID-19 patients in the United Arab Emirates. Oman Med J. ;36(1):e221.

38. Saravi SSS, Beer JH. 2020. Apelin-potential therapy for COVID-19? J Mol Cell Cardiol. 145:84–7.

قياس مستوى ايلابيلا في مصل الدم للكشف المبكر عن إصابة الكلى الحاد في مرضى كوفيد-19 العراقيين الراقدين في المستشفى

الصيدلانية مها حسن غاضي
أ.د. ايمان سعدي صالح

الخلاصة

الخلفية: تسبب فيروس كورونا 2 في جائحة مرض فيروس كورونا 2019 التي اثرت على العالم. تصيب متلازمة التهاب الجهاز التنفسي الشديد الخلية المضيفة باستخدام الإنزيم المحول للأنجيوتنسين 2 كمستقبل له. يعتبر ابلين أو محفزة المستقبلات تأثيرًا وقائيًا في الكوفيد-19من خلال قمع إنتاج الإنزيم المحول للأنجيوتنسين وإنتاج الأنجيوتنسين 2 (Ang-II).

الهدف: هدفت الدراسة إلى تقييم مستوى إيلابيلا في مصل الدم ككاشف مبكر لإصابة الكلى الحاد مع الكوفيد-19.

الطريقة: تضمنت دراسة الاشخاص الاصحاء 43 شخصًا بالغًا كمجموعة تحكم و 45 مريضًا بالغًا تم نقلهم إلى المستشفى في مراكز متعددة في بغداد والتي أظهرت النسخ العكسي الإيجابي في الوقت الحقيقي - تفاعل البوليميراز المتسلسل (RT-PCR) لعينات الجهاز التنفسي التي كانت عبارة عن مسحات أنفية / فموية من (20 - 60) سنة من النتائج السلبية المتضمنة والمستبعدة لـ RT-PCR لـ الكوفيد-19 وحالة الأمراض المصاحبة.

النتائج: أظهرت وجود فروق ذات دلالة إحصائية بين المرضى والمجموعة الضابطة تم أخذها في الاعتبار إحصائيًا عند قيم P <0.05 في eGFR mL / min / 1.73 m2 (قيمة P = 0.000، ELA pg / mL (قيمة P = 0.000، S. الكرياتينين ملغ / مل (قيمة Dimer µg / mL، P = 0.000) (قيمة P = 0.000، NEU × 103 / L (قيمة P = 0.000). ، و LYM × 103 / L (قيمة P = 0.000).

الاستنتاج: ينخفض مستوى ايلابيلا في مصل الدم في المرضى مقارنة مع الأشخاص الأصحاء. يشير مرض الكوفيد-19 إلى أن الأبلين أو مستقبلاته تزيد من تنظيم ACE2 أو تزيد من نشاطها، مما يؤدي إلى قمع نظام ACE-Ang-II الذي يتسبب في إصابة الرئة الحادة في مرضى الكوفيد-19 بحيث يمكن أن يكون ابلين علامة بيولوجية تنبؤية لـ إصابات الرئة وخارج الرئة.

الكلمات المفتاحية: الكوفيد-19، الإنزيم المحول للأنجيوتنسين 2، إصابة الكلى الحاد، إيلابيلا.