The effect of EDTA on people infected with the Corona virus, A review

Wisam M Husien¹

Iraqi Ministry of Education. haditha, Anbar, Iraq (E-mail: wisam.alhaiani@gmail.com)

Abstract: The coronavirus has caused a wave of atypical acute respiratory illness in infected people. This virus has a high transmissibility between humans which has resulted in a worldwide pandemic. It has caused many deaths. No one has found an effective treatment for this problem. Epidemiological studies showed a variation in the severity of the infection among those infected with the virus and looking at the possible explanation for the variation in the severity of the infections. It also showed the involvement of calcium ions in the infection and the effect of the virus on the NLRP3 protein.

Keywords: EDTA, Corona virus, NLRP3, Platelets, Calcium

Introduction

However, the coronavirus, a family of enveloped, single-stranded RNA viruses, that branches into four genera: α -CoVs, β -CoVs, γ -CoVs, and δ -CoVs, only α - and -CoVs have been found capable of on infecting mammals. [1]. Upon binding to their respective receptors, the COVID-19 virus will enter cells by a process of complete endocytosis of viral proteins that results in a mixture of viral inner layers to enable insertion of the viral genome into the cell cytoplasm. [2] The Covid protein is an envelope protein consisting of long amino acids and has two main subunits (S1 and S2) that protrude outside and bind to the enzyme receptor responsible for converting angiotensin 1 to angiotensin 2 (ACE2) [3,4]. The one responsible for binding to the receptor is the terminal amino subunit called the S1 domain. The C terminal, called the S2 ball, contains the structure [5] Virus proteins contain two sites where cleavage occurs, and protease cleavage must occur in order to install S2 in the infected cell membrane. The presence of synthesized cyclic peptides within the S2 subunit. In SARS-CoV-2/COVID-19 of critical importance, the type II transmembrane serine protease TMPRSS2 acts by driving the S1–S2 subunits [6]. It is important to mention here that TMPRSS2 contains two important calcium-binding regions; The SRCR (cysteine-rich scavenger receptor) region (aa 149–242) and the LDLRA (LDL class A receptor) region (aa 113–148) that are the contact site for L-calcium [7] SRCR This region is a conserved calcium center. EDTA acts as a bond breaker due to its ability to chelate calcium [8].

Corona virus relationship with protein NLRP3 and C+2

Viruses encode ion channel proteins called viroporins, and so does the Covid protein. Where these viral proteins act, by certain mechanisms such as the redistribution of ions in the intracellular environment and lysosomal disruption, that lead to the activation of the inflammatory protein NLRP3. The NLRP3 protein recruits the inflammatory protein ASC, which in turn interacts with caspase-1, resulting in its activation. Once activated, caspase-1 will promote the maturation of interleukin (IL)-1 β -6-induced cytokines and this leads to an increase in IL-1 β .) -1 β -6. production. Transport of calcium through the protein ion channel is the key factor in this process, and these findings interestingly correlate between protein ion channel induced ion perturbations at the cell level and the resulting immune pathology exacerbated in a person infected with the viral protein. Protein ion channel activity is specifically associated with severe lung damage, edema accumulation, and then death, and (IL)-1 β -6. The resulting inflammation is associated with these pathological signals. [9,10]

Effect of EDTA on platelets and calcium

In addition to being a cellular effector of blood clotting, platelets are rapidly deployed to infection sites, where inflammatory processes can be modified by interacting with white blood cells, cytokines, cytokines, chemokins, and other inflammatory mediators.[11,12,13,14] Phosphosphateylcholine transfer protein (PCTP) was recently discovered to regulate human platelet aggregation stimulated using PAR4

ISSN NO: 2770-2936

Date of Publication:08-03-2023

ISSN NO: 2770-2936
Date of Publication: 08-03-2023

activated peptide (PAR4AP). However, the role of PCTP after stimulating thrombine, and the mechanisms that PCTP contributes to platelet activation EDTA inhibits PCTP protein Thus it prevents the accumulation

of blood platelets and the formation of blood clots is formed EDTA also reduces Calcium cytoplasmic.[15] **Possible explanation for the difference in severity of infection with COVID-19 among infected people**

The difference in injuries in people Viruses are generally chemically seen as protein. a series of amino acids, and these amino acids are influenced by the median (environment surrounding virus) because it contains two functional groups R-COOH and NH3+ in the acid medium (acid blood) the amino acid is cation and in the base center (basal blood) the amino acid is anion. In the acid medium due to hyper-calcium ions, protein amino acids are associated with calcium ions, where proteins are found to enter the cell only after they are associated with calcium ions. This leads to the increase of virus in the acid medium with hypercalceum and vice versa. In the base center, the amino acid carries negative charge and when it is not hyperetonal calcium, the virus depends on calcium in calcium sites on the cell wall. [16]

Conclusions

From the conclusions obtained, EDTA had multiple actions and was attractive and important as the low level of free calcium ions in the blood indicates that the virus is exclusively bound to calcium ions and enters the cell through the protein ion channel and this replication of the protein on the protein ion channel undoubtedly leads to To the activation of the NLRP3 protein and thus the elevation of the interleukin protein, EDTA had the critical action of reducing the interleukin protein and this indicated that EDTA bound to the free calcium ions and stopped the binding of the virus. With free calcium ions, and thus stops multiplying the ion channel protein and this leads to inhibition of the NLRP3 protein and the resulting deficiency of the inflammatory interleukin protein, or EDTA removes the calcium associated with the virus and leaves it alone outside the cell. That the decrease in calcium ions when infected with the Covid 19 virus undoubtedly indicates a decrease in the content of calcium ions in the blood as a result of their association with the virus and that the value of the test after using EDTA that is returned within the normal level is evidence of disengagement between calcium ions and viruses and evidence of EDTA association with the virus through nitrogen bases. Its ability to inhibit NLRP3 protein. [9,10,17] Certainly, EDTA has bound to calcium on the cell wall in the S1 and S2 regions, and has been shown to be responsible for the fusion and incorporation of virus into the cell [8,18]. EDTA binds to PCTP protein where EDTA-PCTP is formed and thus inhibits it and prevents blood clotting. Inhibition of NLRP3 protein and PCTP protein and the resulting inhibition of inflammatory proteins lead to the return of the value ratio of white blood cells of all kinds to normal levels, which directly helps to re-spread to function excellently [15].

References

- 1. Yin Y, Wunderink RG. MERS, SARS and other coronaviruses as causes of pneumonia. Respirology 2018;23(2):130–7.
- 2. Hoffmann M, Kleine-Weber H, Schroeder S, Kruger N, Herrler T, Erichsen S, et al.SARS-CoV-2 cell entry depends on ACE2 and TMPRSS2 and is blocked by a clinically-proven protease inhibitor. Cell 2020.
- 3. Hoffmann M, Kleine-Weber H, Schroeder S, et al. SARS-CoV-2 cell entry depends on ACE2 and TMPRSS2 and is blocked by a clinically proven protease inhibitor. Cell 2020. https://doi.org/10.1016/j.cell. 2020.02.052.
- 4. Walls AC, Park YJ, Tortorici MA, Wall A, McGuire AT, Veesler D. Structure, function, and antigenicity of the SARS-CoV-2 spike glycoprotein. Cell 2020. https://doi.org/10.1016/j.cell.2020.02.058.
- 5. Wan Y, Shang J, Graham R, Baric RS, Li F. Receptor recognition by the novel coronavirus from Wuhan: an analysis based on decade-long structural studies of SARS coronavirus. J Virol. 2020;94(7).
- 6. Coutard B, Valle C, de Lamballerie X, Canard B, Seidah NG, Decroly E. The spike glycoprotein of the new coronavirus 2019-nCoV contains a furin-like cleavage site absent in CoV of the same clade. Antiviral Res 2020;176:104742.

- 7. Paoloni-Giacobino A, Chen H, Peitsch MC, Rossier C, Antonarakis SE. Cloning of the TMPRSS2 gene, which encodes a novel serine protease with transmembrane, LDLRA, and SRCR domains and maps to 21q22.3. Genomics 1997;44(3):309–20. https://doi.org/10.1006/geno.1997.4845.
- 8. Reichhardt MP, Loimaranta V, Lea SM, Johnson S. Structures of SALSA/DMBT1 SRCR domains reveal the conserve
- 9. J. L. Nieto-torres; C. Verdiá-báguena; J. M. Jimenez-guardeño; J. A. Regla-nava; C. Castaño-rodriguez; R. Fernandez-delgado; J. Torres; V. M. Aguilella; L. Enjuanes (2015). Severe acute respiratory syndrome coronavirus E protein transports calcium ions and activates the NLRP3 inflammasome.
- 10. Shah A(2020) Novel Coronavirus-Induced NLRP3 Inflammasome Activation: A Potential Drug Target in the Treatment of COVID-19. *Front. Immunol.* 11:1021. doi:10.3389/fimmu.2020.01021
- 11. Weyrich AS; Zimmerman GA (2004). "Platelets: signaling cells in the immune continuum". Trends Immunol. 25 (9): 489–95.
- 12. Wagner DD; Burger PC (2003). "Platelets in inflammation and thrombosis". Arterioscler. Thromb. Vasc. Biol. 23 (12): 2131–37.
- 13. Diacovo TG; Puri KD; Warnock RA; Springer TA; et al. (1996). "Platelet-mediated lymphocyte delivery to high endothelial venules". Science. 273 (5272): 252–55.
- 14. Iannacone M; Sitia G; Isogawa M; Marchese P; et al. (2005). "Platelets mediate cytotoxic T lymphocyte-induced liver damage". Nat. Med. 11 (11): 1167–69
- 15. Li, Han; Wang, Bangqin; Ning, Leping; Luo, Yu; Xiang, Shulin (2020). Transient appearance of EDTA dependent pseudothrombocytopenia in a patient with 2019 novel coronavirus pneumonia. Platelets, (), 1–2.
- 16. Rickard, Sharon E.; Thompson, Lilian U. (1997-04). ACS Symposium Series. Washington, DC: American Chemical Society p (294–312)
- 17. Abdoulaye C., Brou K. and Jie C., 2011. Phytic Acid in Cereal Grains: Structure, Healthy or Harmful Ways to Reduce Phytic Acid in Cereal Grains and Their Effects on Nutritional Quality. American Journal of Plant Nutrition and Fertilization Technology, 1: 1-22.
- 18. Holleman, A. F.; Wiberg, E. (2001). *Inorganic Chemistry*. San Diego: Academic Press.