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Original Article

Blood viscosity of COVID-19 patient: a preliminary report

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Abstract: COVID-19 is the important global problem. This new coronavirus can cause several hematological changes. Thrombosis is a problem might occur in COVID-19. In COVID-19, thrombosis might be the result of endothelial dysfunction, platelet activation, increasd blood viscosity, and blood flow disturbance. The change of blood viscosity in the patients with COVID-19 is possible but little mentioned. Here, the authors report on a preliminary data on blood viscosity assessment among 41 COVID-19 patients. According to the estimation, the estimated blood viscosity values are 72.4 ± 18.6 and 18.2 ± 2.2 for high and low shear rates. Based on this preliminary report, the increased blood viscosity is detected in COVID-19 patient and it might be associated with thrombohemostatic problem.

Keywords: COVID-19, blood, viscosity

Introduction

COVID-19 is the important global problem. This infection is caused by SARS CoV2 virus. The infection results in acute febrile illness and severe infection might result in death [1, 2]. Apart from respiratory problem, the patient can manifest non-respiratory problem [3]. This new coronavirus can cause several hematological changes. Thrombosis is a problem that might occur in COVID-19 [4]. In COVID-19, thrombosis might be the result of endothelial dysfunction, platelet activation, hyperviscosity, and blood flow aberration [4].

The abnormal blood viscosity is observable in many viral infections such as viral hepatitis and influenza [5, 6]. The problems might be due to the pathology of hematological system or liver due to the infection. The change of blood viscosity in the patients with COVID-19 is possible but little mentioned. The COVID-19 patient might have hyperviscosiy [7]. The thrombosis might occur in either venous or article vascular system [8]. In the patient with hyperviscosity problem, plasma exchange therapy is indicated [7]. Regarding the pathogenesis, the change of endothelial cell, blood cell and plasma protein in COVID-19 are proposed as possible underlying mechanisms for rheological disorders [8, 9]. The thrombophilia resulted from cytokine surge induced inflammation in COVID-19 is also observed for relationship with hyperviscosity and thrombosis [9]. Although there are some previous relevant published studies, there has never been any report showing the exacted blood viscosity value among COVID-19 patients. Here, the authors report on a preliminary data on blood viscosity assessment among 41 COVID-19 patients.

Materials and methods

Here, the authors present observation on 41 patients with COVID-19 (37 females and 4 males, age between 7 and 74 years). This is a retrospective study on available primary data of the patients. The real-time polymerase chain reaction molecular diagnosis was used for confirmation of infection in all cases. All patients had complete recovery from the illness. For blood viscosity assessment, the standard calculation technique proposed by Duyuler et al. is used [6]. Briefly, the two basic primary laboratory parameters, total protein (TP) and hematocrit (HCT) were included for calculation by mathematical modelling technique. The two included parameters are the main factors affecting blood viscosity and it was previously used in mathematical formula development for estimation of the blood viscosity [10]. Any dataset that are not complete were excluded. The calculation was performed based on both high (208/s) and low (0.5/s)

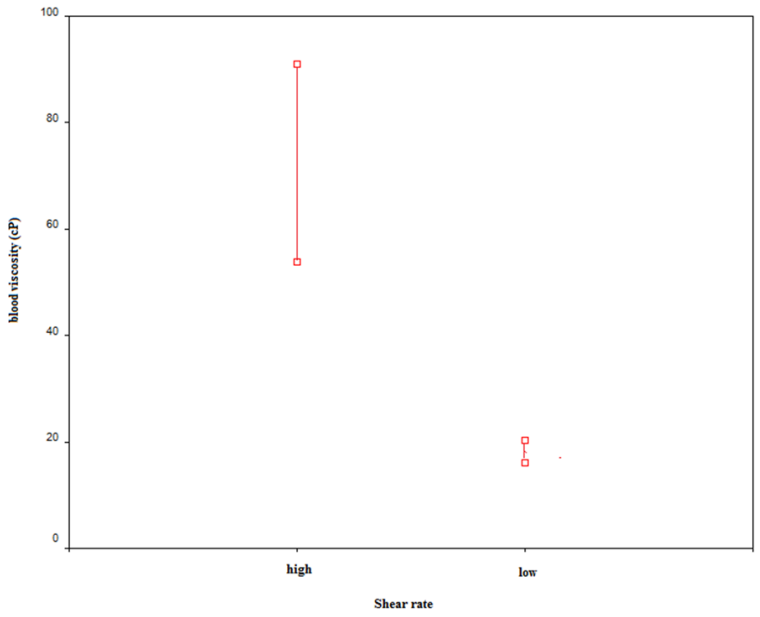

Figure 1. Estimated blood viscosity value in COVID-19.

shear rate conditions, that might occur during blood flow physiological condition [10]. The referenced formula for calculation in both high (208/s) and low (0.5/s) shear rate conditions are "(0.12 × HCT) + 0.17 (TP - 2.07)" and "(1.89 × HCT) + 3.76 (TP - 78.42)", respectively [10]. Regarding the formula, HCT is hematocrit in %, TP is total protein concentration in g/L, and the outcome is the whole blood viscosity in centipoise (cP) [10].

The statistical parameters in calculation are mean and standard deviation. The protocol of this work was approved by local ethical committee (No. SMA1-24/2020). All primary laboratory data are from general medical care of the patients and according to local ethical practice guidelines. This work is a retrospective study and it is a mathematical modeling study that does not deal with human or animal subjects, therefore, the informed consent is not applicable.

Results

According to the estimation, the estimated blood viscosity values are 72.4 ± 18.6 (range 53.8-91) and 18.2 ± 2.2 (range 16-20.4) for high and low shear rates (**Figure 1**). This value is significantly higher than control (56.4 ± 20.9 and 16.9 ± 1.0 for high and low shear rate conditions, respectively). The blood viscosity in COVID-19 group is about 1.98-4.86 times more than control group.

Discussion

Elevated concentrations of acute phase reactants and hypergammaglobulinemia in infection is the cause of blood viscosity change. The increased blood viscosity in acute infections might further cause several clinical problems such as myocardial infarction (MI), venous thrombosis, and venous thromboembolism [10, 11]. The thrombohemostatic disorder is a possible problem in COVID-19 patients. The mechanism that the virus alter normal hemostatic physiology is interesting. In COVID-19, hypoxia, immune reactions, and hypercoagulability might occur and this is an underlying for thrombogenesis process [4]. It is recommended that combine thrombolytic and immunosuppressive therapy is necessary for prevention of COVID-19 related thrombosis [12]. The blood viscosity change is observed in some infections. Regarding respiratory virus infection, the impaired microcirculation and hemodynamics might occur as a result of increased blood viscosity [13]. This phenomenon is well demonstrated in influenza [6]. In COVID-19, the hyperviscosity is possible [7]. A study in the COVID-19 patient with hypercoagulation state show the markedly change of erythrocyte, platelet, serum ferritin, and P-selectin [14].

Nevertheless, there is no systematic assessment on the blood viscosity value. This report is the first report showing the exact value of blood viscosity in COVID-19 patient. The increased blood viscosity is observed. This result can confirm the previous reports that mentioned for the occurrence of clinical problem reflecting hyperviscosity phenomenon in COVID-19 [7]. In this preliminary report, estimated blood viscosity in COVID-19 patient is assessed. It might confirm that there is a change of blood viscosity in COVID-19 patient.

The results in this preliminary study are not enough to draw the conclusion, and more details are needed to be contained. Nevertheless, the data from this preliminary report is useful for further study in thrombohemostatic research in COVID-19. Further studies for

analyzing clinical association of viscosity on seveverity and mortality of COVID-19 are recommended.

Disclosure of conflict of interest

None.

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References

[1] Vieira JM, Ricardo OMP, Hannas CM, Kanadani TCM, Prata TDS and Kanadani FN. What do we know about COVID-19? A review article. Rev Assoc Med Bras (1992) 2020; 66: 534-540.

[2] Xie P, Ma W, Tang H and Liu D. Severe COVID-19: a review of recent progress with a look toward the future. Front Public Health 2020; 8: 189.

[3] Tin SS and Wiwanitkit V. Uncommon atypical presentations of COVID-19: important and should not be under recognized! J Health Sci Med Res 2020; 38: 153-158.

[4] Ahmed S, Zimba O and Gasparyan AY. Thrombosis in coronavirus disease 2019 (COVID-19) through the prism of Virchow's triad. Clin Rheumatol 2020; 39: 2529-2543.

[5] Jang B, Han JW, Sung PS, Jang JW, Bae SH, Choi JY, Cho YI and Yoon SK. Hemorheological alteration in patients clinically diagnosed with chronic liver diseases. J Korean Med Sci 2016; 31: 1943-1948.

[6] Bogomolov BP and Deviatkin AV. Microcirculation and hemostasis in influenza and acute viral respiratory infections complicated with pneumonia. Ter Arkh 2002; 74: 44-8.

[7] Truong AD, Auld SC, Barker NA, Friend S, Wynn AT, Cobb J, Sniecinski RM, Tanksley CL, Polly DM, Gaddh M, Connor M, Nakahara H, Sullivan HC, Kempton C, Guarner J, Duncan A, Josephson CD, Roback JD, Stowell SR and Maier CL. Therapeutic plasma exchange for COVID-19-associated hyperviscosity. Transfusion 2020; 10.1111/trf.16218.

[8] Abouhashem S, Eldawoody H and Taha MM. Cerebral venous sinus thrombosis in patients with COVID-19 infection. Interdiscip Neurosurg 2021; 24: 101091.

[9] Maier CL, Truong AD, Auld SC, Polly DM, Tanksley CL and Duncan A. COVID-19-associated hyperviscosity: a link between inflammation and thrombophilia? Lancet 2020; 395: 1758-1759.

[10] Duyuler PT, Duyuler S, İleri M, Demir M, Dolu AK and Başyiğit F. Evaluation of whole blood viscosity in patients with aortic sclerosis. J Tehran Heart Cent 2017; 12: 6-10.

[11] Sloop GD, De Mast Q, Pop G, Weidman JJ and St Cyr JA. The role of blood viscosity in infectious diseases. Cureus 2020; 12: e7090.

[12] Papamichalis P, Papadogoulas A, Katsiafyl-loudis P, Skoura AL, Papamichalis M, Neou E, Papadopoulos D, Karagiannis S, Zafeiridis T, Babalis D and Komnos A. Combination of thrombolytic and immunosuppressive therapy for coronavirus disease 2019: a case report. Int J Infect Dis 2020; 97: 90-93.

[13] Bogomol'tsev BP and Deviatkin AV. Clinical implications of impaired microcirculation and hemodynamics in acute respiratory viral infections and their pharmacological correction. Klin Med (Mosk) 2003; 81: 9-15.

[14] Venter C, Bezuidenhout JA, Laubscher GJ, Lourens PJ, Steenkamp J, Kell DB and Pretorius E. Erythrocyte, platelet, serum ferritin, and P-selectin pathophysiology implicated in severe hypercoagulation and vascular complications in COVID-19. Int J Mol Sci 2020; 21: 8234.