PROGRESS IN MEDICAL SCIENCES, 2021
VOL 5, NO. 1, PAGE 1 – 3
DOI: doi.org/10.47363/PMS/2021(5)144

CASE REPORT

Open Access

Covid Causing Thrombotic Thrombocytopenic Purpura

Zohra R. Malik[1], James R. Allen[2], Muhammad Mumin[2], Lelesse Mocio[3], Brandon Miller[3] and Zareen Razaq[4]

[1]Corresponding Author. Saint John's Episcopal Hospital, New York, USA

[2]Saint John's Episcopal Hospital

[3]Ross University School of Medicine

[4]Lahore Medical and Dental College

ABSTRACT

COVID-19 has been causing havoc for over a year now. As more cases of COVID have been diagnosed, we are beginning to see various sequelae emerge after patients contract the virus. Specifically, we have seen a patient develop Thrombotic Thrombocytopenic Purpura (TTP) after being diagnosed with COVID-19. Thrombotic thrombocytopenic purpura is a hematologic disorder in which an enzyme deficiency, specifically of metalloprotease ADAMTS-13, causes abnormal multimer formation with the consequences of fever, microangiopathic hemolytic anemia, thrombocytopenia, renal disease, and neurological abnormalities. The coronavirus (COVID-19) has been observed across the world since first emerging in 2019 in China. Although both diseases have been widely observed independently, the development of TTP shortly after contracting COVID has not frequently been reported [1, 2, 3]. Thus, we are bringing to light a case of a patient who developed TTP shortly after being diagnosed with COVID.

ARTICLE HISTORY

Received May 19, 2021

Published May 30, 2021

Introduction

Thrombotic Thrombocytopenic Purpura (TTP) is a rare hematologic disorder in which a person develops a pentad of symptoms including fever, anemia, thrombocytopenia, renal disease, and neurologic abnormalities [4]. The etiology of the disease is related to deficiency of the ADAMTS-13 protease, synthesized in the liver, which normally cleaves vWF into monomers. In its absence, large vWF multimers form and excessive clotting ensues, leading to the sequelae observed in TTP [5, 6]. It was first discovered in 1924; however, it was not until 1997 that it was recognized that a significant contributor to the development of the disease was ADAMTS-13 deficiency [7]. Up until this point, the disease was often fatal. As a result of the identification of this metalloprotease deficiency's relation to the development of TTP, patients who develop the disease can now be treated with supplemental ADAMTS-13 [7].

Criteria for TTP:

- Fever
- Anemia (Microangiopathic hemolytic anemia: schistocytes, elevated LDH, indirect hyperbilirubinemia)
- Thrombocytopenia
- Renal disease
- Neurologic abnormalities

Criteria for COVID-19:

- Positive SARS-CoV-2 RNA PCR Serology [8]

Case Description

We-hereby-present a case of an 82-year-old man with past medical history of hyperlipidemia, benign prostatic hyperplasia, who presented to the ED for hypoxia and altered mental status. Patient was noted to have labored breathing and was desaturating to 70s on non-rebreather which led to intubation. Patient also became hypotensive with a blood pressure of 80/40 and was started on pressor support. Vitals on presentation as shown in table 1.

Table 1: Vitals on Presentation

Blood pressure	80/40 mm Hg
Heart rate	128 beats/min
Temperature	101.7 degree F
Saturation	98% on Ventilator
Respiratory rate	28 breaths/minute

Extensive blood work was done. The most significant labs are shown in the table 2.

Contact Zohra R. Malik ✉ Zohrarazaq@gmail.com Department of Internal Medicine, Saint John's Episcopal Hospital, New York, USA.

© 2021 The Authors. This is an open access article under the terms of the Creative Commons Attribution NonCommercial ShareAlike 4.0 (https://creativecommons.org/licenses/by-nc-sa/4.0/).

Table 2: Labs on Presentation

	Patient's Value	Reference Range
Hemoglobin	16.4 g/dl	12.0-18.0 g/dl
Hematocrit	57.2 %	37-47 %
Mean Corpuscular Volume	92 FL	81-99 FL
White Blood Cell Count	14.9 x 10^3/uL	5.2-12.4 x 10^3/uL
Platelet Count	203 x 10^3/uL	130-400 x 10^3/uL
Sodium	181 mmol/L	137-145 mmol/L
Potassium	3.9 mmol/L	3.5-5.1 mmol/L
Bicarbonate	19 mmol/L	22-30 mmol/L
BUN	82.0 mg/dl	7.0-17.0 mg/dl
Creatinine	4.00 mg/dl	0.52-1.04 mg/dl
Lactic acid	7.7 mmol/L	0.7-2.1 mmol/L
Ferritin	546 ng/ml	17.9-464 ng/ml
Lactate Dehydrogenase	1130 U/L	313-618 U/L
D-Dimer	2382 ng/ml	0-229 ng/ml

SARS-CoV-2 Rap RNA came back positive. Patient was admitted to ICU for respiratory failure secondary to Covid Pneumonia with superimposed bacterial pneumonia. Patient was treated with broad spectrum antibiotic and intravenous fluids. Peripheral blood film results as shown in table 3.

Toxic Granulation	Present	
Platelet Estimate	Decreased	
Large Platelets	Present	
Giant Platelets	Present	
Schistocytes	1+	
Haptoglobin	<20 mg/dl	34-200 mg/dl

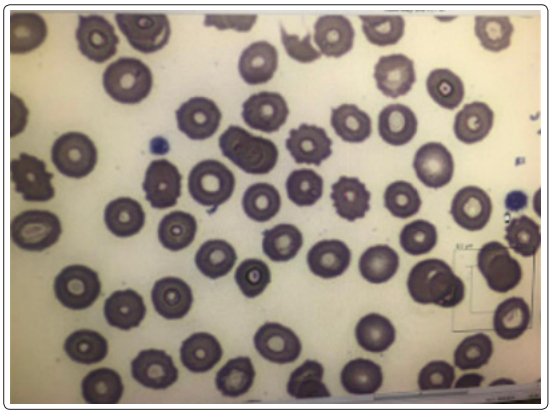

Figure 1: Showing schistocytes

Patient developed thrombocytopenia, microangiopathic hemolytic anemia. Peripheral blood smear showed schistocytes and low haptoglobin. Heparin induced thrombocytopenia antobodies came back negative. Patient had renal function abnormalities on presentation. Thus completing our picture of TTP. Over time patient condition improved with IV antibiotic and IV fluids. Patient was back to his baseline, given an out-patient hematology appointment and discharged to nursing home.

Case Discussion

The novel coronavirus was identified as the cause of a pneumonia-like disease towards the end of 2019 in Wuhan, China. In February 2020, the World Health Organization (WHO) designated this disease as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) or COVID-19. Since then, the spread has been rapid worldwide with over 160 million cases and over 3.3 million deaths to date. Our understanding of COVID-19 is ever-changing with new cases and studies that emerge. The spectrum of disease ranges from mild to critical, with 81 percent categorized as a mild disease[9]. Patients typically develop symptoms within 14 days of exposure[9]. Cohort studies have shown that common symptoms include cough, fever, myalgia, headache, dyspnea, sore throat, and diarrhea. Studies began to show that hematologic complications, most notably thromboembolisms, were prominent[10, 11]. This led to the recommendation of starting patients on anticoagulation who are hypoxic. As time moved forward, physicians and scientists' understanding of this deadly disease evolved. More studies and case reports emerged showing a multitude of conditions that may be associated with COVID-19. One such disease was TTP. Not much is known about how Covid-19 affects the diagnosis and treatment of TTP. Standard TTP therapies including plasma exchange therapy should be instituted. Though there were initial reports of corticosteroids increasing the risk of more severe covid, recent data has suggested that dexamethasone may improve outcomes in COVID-19 patients who are hypoxic [12]. Due to these findings, steroids should be initiated early in patients with COVID-19 induced TTP. Rituximab is a drug used for TTP that targets CD20 antigen. Rituximab has been shown to be associated with more severe COVID-19 thus should be used with caution [13].

Conclusion

As COVID-19 cases are diagnosed, we have noticed a connection to another condition. In this case report, we have shown support for the association between COVID-19 and TTP. A COVID-19 patient may develop TTP. A person may present with the criteria for TTP (Fever, Anemia, Thrombocytopenia, Renal Disease and Neurological abnormalities) in the presence of COVID-19. Our case report demonstrates a person who satisfies these criteria with COVID-19 as a contributing factor.

References

[1] Altowyan E, Alnujeidi O, Alhujilan A, Mohammed A. COVID-19 presenting as thrombotic thrombocytopenic purpura (TTP). BMJ Case Reports CP 2020; 13:238026.

[2] Marco Capecchi, Cristina Mocellin, Chiara Abbruzzese, Ilaria Mancini, Daniele Prati, et al. Dramatic presentation of acquired thombotic thrombocytopenic purpura associated with COVID-19. Haematologica 2020; 105:540.

[3] Hamed AzhdariTehrani, MaryamDarnahal, MohammadVaezi, ShirinHaghighi. "COVID-19 Associated Thrombotic Thrombocytopenic Purpura (TTP); A Case Series and Mini-Review." International Immunopharmacology, Elsevier 2021, www.sciencedirect.com/science/article/pii/S1567576921000333.

[4] "Thrombotic Thrombocytopenic Purpura." National Heart Lung and Blood Institute, U.S. Department of Health and Human Services, www.nhlbi.nih.gov/health-topics/

thrombotic-thrombocytopenic-purpura.

[5] Theodore Wun, MD. "Thrombotic Thrombocytopenic Purpura (TTP)." Practice Essentials, Background, Pathophysiology, Medscape, 3 Apr. 2021, emedicine.medscape.com/article/206598-overview.

[6] Zheng XL. ADAMTS13 and von Willebrand factor in thrombotic thrombocytopenic purpura. Annual review of medicine 2015; 66:211–225.

[7] Lämmle B, Kremer Hovinga JA, Alberio L. Thrombotic thrombocytopenic purpura. J Thromb Haemost 2005; 3:1663-1675.

[8] Caliendo, Angela M, Kimberly E Hanson. Edited by Martin S Hirsch and Allyson Bloom, COVID-19: Diagnosis 2021, www.uptodate.com/contents/coronavirus-disease-2019-covid-19-diagnosis?topicRef=127454&source=see_link.

[9] Chan JF, Yuan S, Kok KH. A familial cluster of pneumonia associated with the 2019 novel coronavirus indicating person-to-person transmission: a study of a family cluster. Lancet. 2020; 395:514-523.

[10] Klok FA, Kruip MJHA, van der Meer NJM. Incidence of thrombotic complications in critically ill ICU patients with COVID-19. Thromb Res 2020; 19:145-147.

[11] Bilaloglu S, Aphinyanaphongs Y, Jones S, Iturrate E, Hochman J, et al. Thrombosis in Hospitalized Patients With COVID-19 in a New York City Health System. JAMA 2020; 324:799-801.

[12] Catalans S, Coppo P, Scully M. COVID-19 and thrombotic thrombocytopenia purpura: frequently asked questions. American Society of Hematology. http://www.hematology.org/covid-19/covid-10-and-ttp(Accessed 17 May 2021)

[13] Avouac J, Drumez E, Hachulla E, Raphaèle S, Sophie G-L. COVID-19 outcomes in patients with inflammatory rheumatic and musculoskeletal diseases treated with rituximab: a cohort study [published online ahead of print, 2021 Mar 25]. Lancet Rheumatol 2021;10.1016/S2665-9913(21)00059-X.