



Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.

Critical Care

SESSION TITLE: Medical Student/Resident Critical Care Posters

SESSION TYPE: Med Student/Res Case Rep Postr

PRESENTED ON: October 18-21, 2020

DOUBLE TROUBLE: A CASE OF SUSPECTED ANTIPHOSPHOLIPID SYNDROME IN THE SETTING OF HYPERCOAGULABLE COVID-19 PNEUMONIA

SARAH UPSON MICHAELA MURPHY FARAH ASHRAF AND SALEEM SHAHZAD

INTRODUCTION: The hypercoagulable state seen in Covid-19 patients has been well described. Our case of a patient with limb ischemia and elevated Antiphospholipid Syndrome (APS) antibodies raises the question of multiple mechanisms of hypercoagulability with Covid-19 infection.

CASE PRESENTATION: 61 year old female with fibromyalgia and osteoarthritis presented with dyspnea and cough. On arrival, she was tachypneic (38 breaths/min) with low oxygen saturation (91%). Chest radiograph showed vascular congestion with bilateral interstitial infiltrates. She tested positive for SARS-CoV2 19 and was started on hydroxychloroquine, ceftriaxone, and azithromycin. She was intubated day 2 and was transferred to ICU, where she required vasopressor support for septic shock. She developed transient thrombocytopenia, which was attributed to sepsis, as Heparin-Induced Thrombocytopenia was ruled out. Her fingertips were cyanotic and toes gangrenous, concerning for limb ischemia. Given her instability, she was not a candidate for vascular intervention. Further workup revealed positive APS antibodies (Table 1) and therapeutic dose lovenox was started. Hospital course was further complicated by polymicrobial pneumonia with Computed Tomography Chest showing bilateral cavitations and traction bronchiectasis. Despite maximal therapy, she expired on day 33 of hospitalization.

DISCUSSION: The inclusion of APS antibodies in the ever-evolving body of knowledge regarding Covid-19 may play a crucial role in uncovering an additional mechanism of thrombosis to its already hypercoagulable state. The current literature on Covid-19 suggests direct endothelial injury and complement activation as possible sources of thrombosis. In this case the workup for limb ischemia revealed elevated anti-B2-Glycoprotein 1 IgA and IgM antibodies, anti-Phosphatidylserine IgM and IgG antibodies, and anti-Cardiolipin IgA and IgM antibodies. Although infections have been shown to induce transient elevations in APS antibodies, not necessarily associated with thrombosis, we put forth that our patient's multiple high titers of IgM and IgG antibodies in the clinical setting of acute thrombosis supports potential superimposed APS. Unfortunately, the patient expired before repeat antibody testing could be completed in congruence with a definitive diagnosis of APS.

CONCLUSIONS: This case highlights the need for research regarding the mechanism in which Covid-19 may potentiate Antiphospholipid Syndrome amidst an already thrombotic disease state. Such research would promote the need for early APS antibody testing and initiation of anticoagulation in the amplified hypercoagulable state in Covid-19 with APS, especially in the prevention of life threatening thrombosis.

Reference #1: Magro C, Mulvey JJ, Berlin D, et al. Complement Associated Microvascular Injury and Thrombosis in the Pathogenesis of Severe COVID-19 Infection: a Report of Five Cases. *Transl Res.* 2020;S1931-5244(20)30070-0. doi:10.1016/j.trsl.2020.04.007

Reference #2: McDonnell T, Artim-Esen B, Wincup C, Ripoll VM, Isenberg D, Giles IP, Rahman A, Pericleous C. Antiphospholipid Antibodies to Domain I of Beta-2-Glycoprotein I Show Different Subclass Predominance in Comparison to Antibodies to Whole Beta-2-glycoprotein I. *Front. Immunol.* 2018; 9 (2244): 1-7. DOI: 10.3389/fimmu.2018.02244

Reference #3: Nakayama T, Akahoshi M, Irino K, Kimoto Y, Arinobu Y, Niuro H, Tsukamoto H, Horiuchi T, Akashi K. Transient Antiphospholipid Syndrome Associated with Primary Cytomegalovirus Infection: A Case Report and Literature Review. *Case Reports in Rheumatology.* 2014; 2014 (271548): 1-6. DOI: 10.1155/2014/271548

DISCLOSURES: No relevant relationships by Farah Ashraf, source=Web Response

No relevant relationships by Michaela Murphy, source=Web Response

No relevant relationships by Saleem Shahzad, source=Web Response

No relevant relationships by Sarah Upson, source=Web Response

DOI: <http://dx.doi.org/10.1016/j.chest.2020.08.801>

Copyright © 2020 American College of Chest Physicians. Published by Elsevier Inc. All rights reserved.