

Case Report

A Case of Immune Thrombocytopenia in an Elderly Vaccinated Patient Following COVID-19 Infection

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We report on an elderly fully vaccinated patient with coronavirus 19 (COVID-19) who contracted severe thrombocytopenia and responded well to high-dose prednisone and intravenous immunoglobulin. This is perhaps the first reported case in the literature of the development of profound thrombocytopenia in a fully vaccinated COVID-19 patient. The favorable outcome in this patient may be attributed to his earlier full vaccination against COVID-19.

Keywords: Covid-19 infection; thrombocytopenia**Introduction**

Immune thrombocytopenia is rare and is considered to be an autoimmune disease characterized by isolated thrombocytopenia. Patients may be asymptomatic at presentation or they may present with mild mucocutaneous to a life-threatening bleeding situation [1]. The etiology is diverse and includes conditions like primary immune thrombocytopenic purpura in which the exact cause is unknown or it can be drug-induced, associated with lymphoproliferative disorders, or immunodeficiency syndrome, post infection (mainly viral), and other autoimmune diseases. Mild thrombocytopenia (platelet counts of 100-150 X 10⁹/L) can be observed in novel coronavirus (COVID-19) disease, however severe thrombocytopenia is rare [2]. We here report a case of severe thrombocytopenia in an elderly patient who was fully vaccinated six months prior to his presentation and who has responded well to high dose prednisone and intravenous immunoglobulin.

Case Report

The patient is a 93 years old white male with a past medical history significant for coronary arterial disease status-post coronary artery bypass graft, hypertension, hypothyroidism, chronic kidney disease stage III, and atrial flutter who was fully vaccinated six months before his admission and went to the Emergency Room (ER) at a local hospital with four days history of fatigue, fever, and dry cough. An oropharyngeal swab for COVID-19 testing was positive and the patient was diagnosed with acute hypoxic respiratory failure due to COVID-19 infection. He was then treated with oxygen and dexamethasone but not with remdesivir because of his chronic kidney disorder. He was off oxygen before his discharge seven days later but required readmission two days after for the superimposed bacterial pneumonia and received 7 days course of intravenous antibiotic (Zosyn). Once stabilized he was discharged for rehabilitation to a nursing home. However, while in the nursing home 14 days later he developed marked thrombocytopenia and was sent back to the emergency room for further evaluation.

At ER he was febrile with a temperature of 101.8 F and had a respiratory rate of 30 breaths per minute and oxygen saturation of 88-89% while he was breathing ambient air. Breath sounds

were diminished bilaterally with bibasilar rales. The abdominal examination was normal. There were no bruises, bleeding, purpuric rash, or ecchymosis.

Laboratory investigations showed WBC 7.7 X 10⁹/L, hemoglobin 10.6 g/dL and platelet count 17 X 10⁹/L. Prothrombin time, activated partial thromboplastin time and fibrinogen levels were normal. A peripheral blood smear showed thrombocytopenia and occasional schistocytes. Vitamin B12 and folate levels were obtained and found to be normal. Antiplatelet factor 4 and antiplatelet antibodies were not detected. Liver function tests were normal. His BUN was normal at 19 mg/dL but creatinine was slightly high at 1.77 mg/dL and GFR was low at 44 ml/min. A chest Computed Tomography (CT) showed ground-glass opacities in the lower zones. Although antiplatelet antibodies were negative, the temporal sequence suggested that COVID-19 was the causal agent of immune thrombocytopenia in this patient.

The patient was admitted to the hospital and began to receive treatment with intravenous antibiotics, low molecular weight heparin subcutaneously and oxygen via nasal cannula. He was transfused with one unit super-pack platelet and also received high-dose intravenous methylprednisolone (125mg every 6 hours) with Protonix 40 mg intravenously once a day. Two days later as his platelet count failed to show any improvement, Intravenous Immunoglobulin (IVIg) was administered at a rate of 1g per kilogram of body weight daily. Following 2 doses of IVIg, his platelet counts began to rise and on the fourth day of hospitalization his CBC revealed WBC 10.7 X 10⁹/L, hemoglobin 10.6 g/dL, and platelet count 96 X 10⁹/L. On the 6th day, he was discharged on oral prednisone (60 mg per oral daily) to a skilled nursing facility where he continued to recover. Although he remained anemic (Hb 10.4 g/dl) and thrombocytopenic (platelet 104 x10⁹/L) but in a stable condition with no bruising, bleeding, or purpuric rash. Bone marrow aspiration and biopsy were contemplated when platelet count did not show any improvement following IV steroid therapy but were not performed due to the fear of possible hematoma development.

Discussion

COVID-19 is known to cause severe respiratory symptoms and

complications but, available data also suggest that the symptoms of the disease can also result from the involvement of other body systems including hematopoietic, neurological, and the immune system [3,4]. In line with recent reports, it is reasonable to postulate that the profound thrombocytopenia, in this case was, COVID-19 induced. Indeed, the emergence of immune thrombocytopenia in the context of COVID-19 is increasingly reported [5-9]. Although a causal link between COVID-19 and immune thrombocytopenia has not yet been firmly established, it is suggested by the temporal association with the COVID-19 pandemic and the history of exposure of affected patients to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [10].

Recent publications suggest poor outcomes in patients with covid-19 disease associated with thrombocytopenia [11]. However, no information is available regarding the association of COVID-19 infection in fully vaccinated COVID-19 patients with thrombocytopenia and its outcome. Our patient was fully vaccinated with two doses of Pfizer's vaccine six months before his contracting COVID-19 infection. Although the patient is 93 years old, to our surprise, he responded well to conventional therapy for immune thrombocytopenia. Although the current data suggest poor prognosis in COVID-19 with thrombocytopenia. We believe the described patient's favorable outcome was attributable to his prior vaccination against COVID-19 infection.

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