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Immune Thrombocytopenic Purpura (ITP) As a Complication of COVID-19: A Case Report

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ABSTRACT

Immune thrombocytopenic purpura (ITP) is a condition that can occur either spontaneously or as a secondary complication of preexisting disorders, often associated with viral infections. In the context of COVID-19, hematological manifestations, including thrombocytopenia, have been observed and linked to increased mortality rates among severely infected patients.

Presented here is a case study of a 70-year-old man who recovered from COVID-19 but subsequently developed ITP. The severity of his thrombocytopenia exceeded what is typically observed during COVID-19 infections, leading to a diagnosis of ITP. Treatment with steroids and intravenous immunoglobulin (IVIG) was initiated based on specialist recommendations, resulting in an adequate response.

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Introduction

A range of hematologic issues have been documented in individuals afflicted with SARS-CoV-2, the coronavirus responsible for COVID-19 infection. The presence of a hypercoagulable condition gives rise to thrombotic complications in patients affected by COVID-19 [1]. However, thrombocytopenia affects approximately one-third of COVID-19 patients and up to two-thirds in cases of severe disease [2]. Thrombocytopenia was predominantly mild and reversible during hospitalization in cases of SARS-CoV-1-associated infection [3]. The immune response to viral infection can result in a reduction of platelet production in the bone marrow and the generation of autoantibodies [3].

Immune thrombocytopenic purpura (ITP), defined by a platelet count < 100 × 10^9/L, classically presents with mucocutaneous bleeding (4), has also appeared as a complication of COVID-19 [3, 5-7]. ITP can manifest either spontaneously or may be secondary to preexisting disorders, such as autoimmune and collagen vascular diseases, lymphoproliferative disorders, and viral infections caused by human immunodeficiency virus (HIV), varicella-zoster virus (VZV), Epstein-Barr virus (EBV), cytomegalovirus

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(CMV), herpes simplex viruses (HSV), hepatitis C virus (HCV), parvovirus, rubella, measles, and more recently, following Zika virus infection [2, 4].

Presented here is a case of a 70-year-old man who recovered from COVID-19 and then developed ITP.

Case presentation

A 70-year-old man with no history of COVID-19 vaccination presented to the emergency room with petechial lesions on his limbs. The patient had experienced mild COVID-19 symptoms, including fever, myalgia, and weakness, two weeks prior to admission. A nasopharyngeal COVID-19 Polymerase chain reaction (PCR) test was positive, and the patient had been exposed to a confirmed COVID-19 patient 20 days prior. The patient's medications included sertraline, tamsulosin, and favipiravir. On admission, the patient was afebrile with normal vital signs and an oxygen saturation of 96%. Purpuric eruptions were observed on the limbs, but there were no signs of bleeding.

Laboratory evaluation revealed severe thrombocytopenia, with a platelet count of 10,000 /mm3. The patient's white blood cell count (WBC), c-reactive protein (CRP), erythrocyte sedimentation rate (ESR), hemoglobin (HB), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) levels were also abnormal. Immunology assay results indicated normal complement components (c3, c4) and negative antinuclear antibody (ANA), anti-neutrophil cytoplasmic antibody (ANCA), anti-double-stranded DNA (Anti ds-DNA), and anticardiolipin Ab. Peripheral blood smear (PBS) revealed low platelet counts, without schistocytes.

Laboratory tests, including coagulation studies, lactate dehydrogenase (LDH) levels, and haptoglobin levels, were performed to help rule out disseminated intravascular coagulation (DIC) and thrombotic thrombocytopenic purpura (TTP). Finally, imaging studies, such as CT scans, were used to evaluate for any signs of organ involvement which do not suggest a diagnosis of TTP or DIC.

A diagnosis of COVID-19-associated ITP was made after excluding other causes of thrombocytopenia, including DIC, bacterial sepsis, and medications. The patient was started on intravenous immunoglobulin (IVIG) at 30 g/daily and methylprednisolone at 1g/daily for three days, along with prednisolone at 50 mg. The patient's platelet counts increased to 77,000 /mm^3 on the fifth day of treatment, and the initial response to glucocorticoid and intravenous immunoglobulin was positive. The patient's platelet counts at discharge were 113,000 /mm^3.

Discussion

ITP is a bleeding disorder caused by an autoimmune response, characterized by a platelet count of less than 100×10^9/L. It typically presents with petechiae or purpuric eruptions [4]. In some instances, ITP has been observed as a complication of COVID-19 [3, 5-7].

ITP involves platelet destruction and inhibition of platelet production. Viruses can infect megakaryocytes, leading to decreased platelet production [8]. A case was encountered of a 70-year-old man who presented with petechial lesions on his limbs but had no active bleeding. A systematic review [3] explored newly diagnosed ITP cases associated with COVID-19 up until August 25, 2020. The review revealed that 71% of cases occurred in individuals over 50 years of age. Interestingly, 7% of asymptomatic COVID-19 patients also developed ITP. Therefore, in cases of newly diagnosed ITP, regardless of COVID-19 symptoms, it is essential to conduct COVID-19 testing, particularly during the ongoing global pandemic [3, 8]. Bomhof et al. reported a mortality case of COVID-19-associated immune thrombocytopenia resulting from intracranial hemorrhage (5, 9). It is worth noting that ITP can develop not only during the active phase of COVID-19 but also after the resolution of COVID-19 symptoms leading to the diagnosis of ITP induced by the virus after ruling out other causes of thrombocytopenia. Additionally, rare cases of vaccine-induced immune thrombocytopenia have been reported globally. Schultz et al. presented five cases of vaccine-related venous thromboembolism occurring 7 to 10 days after vaccination, accompanied by thrombocytopenia [10, 11]. Previous research has identified several potential risk factors for ITP, including a history of previous ITP episodes, chemotherapy, immunotherapy, and immunosuppressive drugs [12].

However, in this case, no such risk factors were identified in the patient's medical history. The primary goal in treating ITP is to increase platelet counts to a level that prevents major bleeding rather than aiming for a return to normal platelet ranges. Response definition is a platelet number between 30 and 100×10^9/L or at least doubling of the baseline count [13]. Prompt diagnosis and treatment of ITP as a complication of COVID-19 infection is imperative, because delayed recognition may result in mortality [5]. Death was reported in a 67-year-old man with ITP associated with COVID-19 because of intracranial hemorrhage within 24 hours [5]. In a

systematic review, a short course of glucocorticoids and intravenous immunoglobulin had a good initial response except for one case with a delayed lag response [3, 14]. Promptly considering ITP following a COVID-19 infection is crucial, as it has the potential to become a serious complication.

Conclusion

ITP can indeed present as a complication of COVID-19, with cases reported during both the active phase of the infection and after the resolution of COVID-19 symptoms. A comprehensive understanding of the relationship between ITP and COVID-19 is crucial to deliver effective and timely care to affected patients. Although certain risk factors have been identified, such as a history of previous ITP episodes and exposure to chemotherapy, immunotherapy, or immunosuppressive drugs, it is worth noting that ITP can also occur in the absence of these factors. Therefore, the presence of petechiae during and after a COVID-19 infection should be recognized as a significant indicator of potential ITP development.

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Ethical Considerations

Compliance with ethical guidelines

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Conflict of Interests

The authors have no conflict of interest to declare.

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