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Severe immune thrombocytopenia in a patient recovering from COVID 19

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Abstract *:*
COVID-19 is a systemic infection with a significant impact on the hematopoietic system and hemostasis. Mild thrombocytopenia has been shown to occur in patients with COVID-19, usually noted on admission to hospital. We report a case of a severe thrombocytopenia in a patient in the recovering stage of COVID-19. We conclude that an immune mechanism was responsible as common causes were excluded. The patient were successfully treated with intravenous immunoglobulins and steroids. This report highlights the need for vigilant monitoring for complications associated with COVID-19.

Keywords*: Immune thrombocytopenic purpura, thrombocytopenia, COVID-19, Intravenous immunoglobulins.*

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I. Introduction :

Coronavirus disease 2019 is a multisystemic disorder caused by severe acute respiratory syndrome. Hematological changes are common in patients with COVID-19, wich include lymphopenia and thrombocytopenia and hypercoagulopathy[1]. Mild thrombocytopenia has been associated with severe COVID-19 disease [2], and usually noted on admission to hospital. Severe thrombocytopenia is rarely reported in COVID-19 patients. We report a case of a severe thrombocytopenia in a patient in the recovering stage of COVID-19.

Patient and observation :

A 53-year-old man with gout under allopurinol, presented to the emergency department with a 3 days history of cough, dyspnea followed by fever and myalgias. A recent trip abroad has not been reported. The initial physical examination revealed a body temperature of 38.7°c, respiratory rate of 17 breaths par minte, heart rate of 90 bpm and oxygene saturation of 97% on ambiant air. Lung auscultation was normal. A chest CT scan showed multiple ground glass in the lower zones. A blood test showed a normal leukocyte count (8.2×103 uL) with lymphopenia (0.8 $\times 10^3$ uL), normal hemoglobin count (13 g/dl) and a normal platelet count (165$\times 10^3$ uL). All other laboratory test (C-reactive protein, lactate deshdrogenase, liver fonctions, d-dimeres and fibrinogene) were withen the normal range. Respiratory viral panel PCR was positive for coronavirus. The patient was diagnosted with moderate COVID19 and received 600mg of hydroxychloroquine daily , azithromycin and preventive low weight heparin. The patient's symptoms improved, laboratory tests remained normal. However on day 20, physical examination revealed signs of gum bleeding. Examination of the skin was remarkable for petechiae over the chest and limbs (figure1). A thorough clinical examination was then performed. A blood test showed a decreased platelet count of 5×10^3 uL. A peripheral blood smear was normal. Autoimmune work-up including ENA and ANCA serology was negative. A bone marrow smear showed abundant cell lines and no haematophagocytosis. There were no anti-PF4 heparin antibodies. Consumption coagulopathy and haemolysis were excluded. The patient received a single infusion of intravenous immunoglobulins (IVIG) : 1 g/kg of body weight and dexamethasone 40 mg for 4 days. His platelet count progressively normalized and was stable 1 month later (figure2).

II. Discussion :

Immune thrombocytopenic purpura (ITP) is an autoimmune systemic disease characterized by the presence of low blood platelets count ($<10^5$/ml) and the production of autoantibodies

against glycoproteins expressed on the platelet surface. An increasing number of studies suggest an association between infectious agents and ITP onset [3]. Many viruses have been identified as triggering the autoimmune process, including HIV, HCV, CMV, EBV, herpes viruses [4, 5]. Thrombocytopenia is detected in 5–41.7% of COVID-19 patients [4, 6,7], and it is typically mild. Severe thrombocytopenia is only rarely

reported in COVID-19 patients, for instance, in association with an immune thrombocytopenic purpura-like state [8, 9].

Diagnosis of COVID-19-associated ITP may be difficult because of several other potential causes, for instance the coagulation activation by COVID-19 infection leading to disseminated intravascular coagulopathy and subsequent thrombocytopenia. Also, treatment of COVID-19, including heparin, azithromycin and hydroxychloroquine, may lead to thrombocytopenia[6]. Glucocorticoids comprise the primary treatment of ITP. Treatment with IVIG in an early stage of COVID-19 may be successful in treatment of COVID-19 infection [10]. Additionally, some COVID-19 patients who suffered deterioration of clinical symptoms have been salvaged by IVIG treatment [11]. IVIG was chosen in our patient because of active bleeding with a good response.

III. Conclusion :

Covid 19 can cause profound and symptomatic thrombocytopenia. An immune aetiology should be considered when other mechanisms have been ruled out in profound and acute thrombocytopenia. The goal of ITP treatment is preventing severe bleeding by providing a safe platelet count. Intravenous immunoglobulin are very effective treatments for severe ITP.

Competing interests :
The authors declare no competing interests.
Contribution of authors :
All authrs have contributed to this work. All the authors have read and agreed to the final manuscript.

Figures :
Figure 1 : Petechies in the lower limbs.
Figure 2 : The course of platelet counts of patient with Covid-19-associeted ITP.

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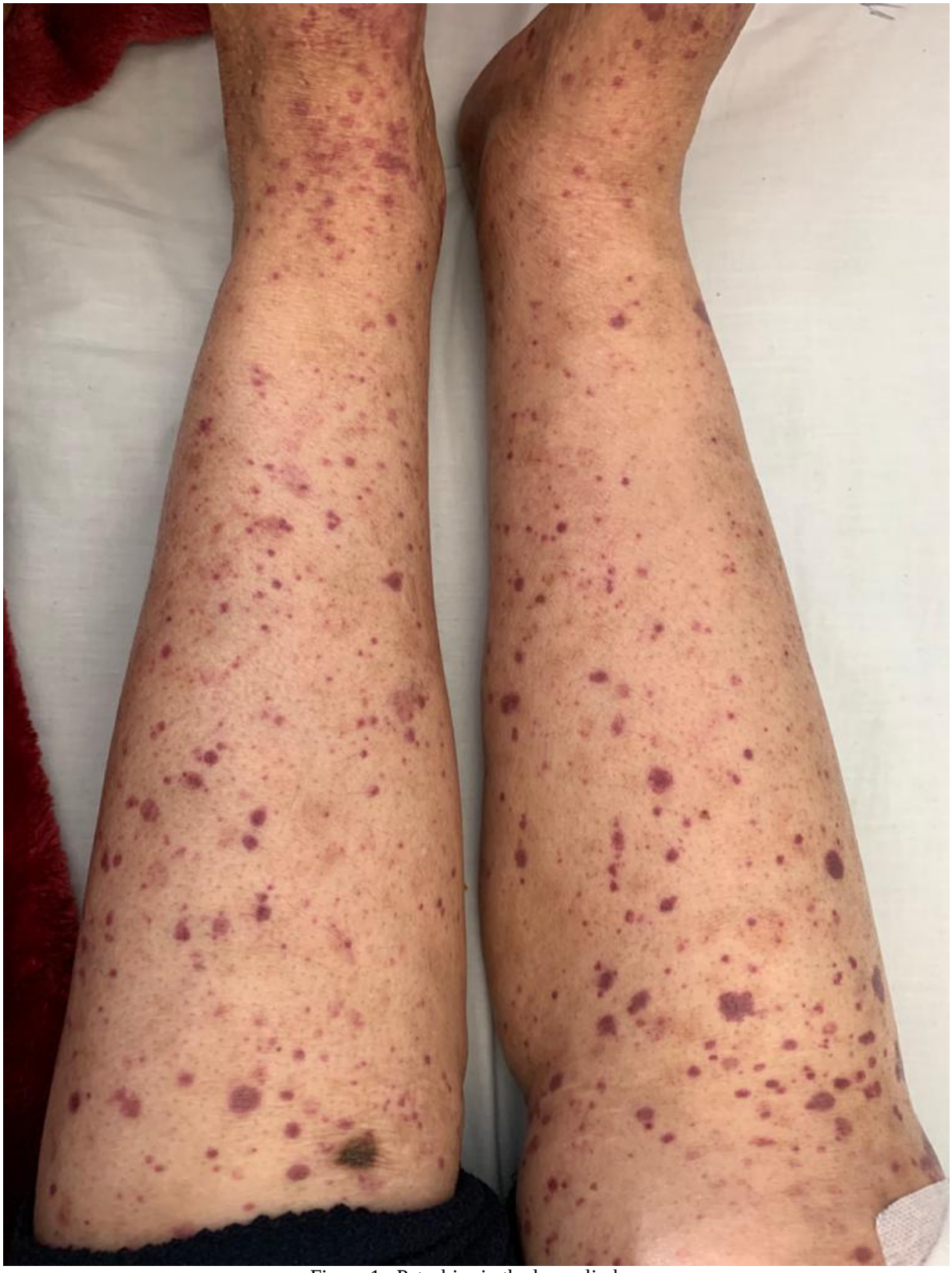

Figure 1 : Petechies in the lower limbs.

Figure 2 : The course of platelet counts of patient with Covid-19-associeted ITP.

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