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Secondary Thrombocytosis Due to Inflammatory Response in SARS-COV2 Infection in Two Blood Related Patients

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Abstract

SARS-COV 2 infection is usually associated with platelet count alterations. In most cases, platelet dysregulation manifests with mild to severe thrombocytopenia. We report the cases of two patients, blood related, who were hospitalized in our department due to SARS-COV2 infection accompanied by mild to severe thrombocytosis. Thrombocytosis is a rare manifestation in COVID-19 patients. Follow-up tests demonstrated a quick return of platelets within normal range, making the diagnosis of secondary thrombocytosis due to SARS-COV 2 infection the most likely diagnosis. Moreover, the blood relation between the two patients creates a suspicion that excessive platelet reaction could be due to genetic predisposition.

Keywords: SARS-COV2 • Thrombocytosis • Platelet count

Introduction

Infection from SARS-COV2 is usually associated with thrombocytopenia. Thrombocytopenia is the main alteration observed and its correlation with increased mortality is well documented [1,2]. Low platelet count seems to be an independent risk factor for severe illness. In a meta-analysis of nine studies which compared platelet count in severe and non-severe cases, there was a significantly lower platelet count in the severe illness group [2]. The mechanisms that may explain thrombocytopenia in COVID-19 patients include decreased platelet production caused by lung injury, potential bone marrow involvement and increased platelet consumption due to immune system activation and microthrombi creation [3]. It is believed that the lungs play a crucial role in platelets biogenesis [4]. On the other hand, thrombocytosis is a rare manifestation of SARS-COV2 infection with, potentially, distinct clinical features and overall survival rate. Prognosis in patients with thrombocytosis seems to be better in comparison with normal as well as with low platelet count groups [5].

Case Presentation

Two sisters, 61 and 72 years old, came to our E.D. with dyspnea, tachypnea and cough. Both had complementary lung sounds (fine crackles in both lung bases) without any other abnormal findings upon clinical examination. The 61 year old lady presented with low partial oxygen pressure in blood gasses (PO_2=63 mmHg) and was thus supported with low O_2 supply by nasal cannula at 5 Lt/min. The 72 year old lady presented with borderline partial oxygen supply (PO_2=83.3 mmHg) and was thus supported with nasal cannula at 2 Lt/min. Their personal medical history contained, in both cases, diabetes mellitus under medication with metformin. The 72 year old lady had a history of transient ischemic attack for which she was under treatment with an antiplatelet agent (acetylsalicylic acid), hypertension under medication with olmesartan/hydrochlorothiazide and hyperlipidemia treated with a combination of atorvastatin/ezetimibe. On the other hand, the 61 year old lady had a history of depression with psychotic features treated with escitalopram, paliperidone and olanzapine, hyperlipidemia treated with simvastatin and hypothyroidism treated with levothyroxine of 100 mg per day. The chest CT scan showed, in both cases, ground-glass opacities with bilateral distribution without any other significant findings.

The laboratory tests demonstrated mild microcytic anemia in both cases due to known thalassemia trait, increased inflammatory markers and moderate to severe thrombocytosis (609 × $10^3/\mu L$ and 901 × $10^3/\mu L$). Thrombocytosis was not previously observed in their medical history. Both patients were treated with the established protocol for COVID-19 (Remdesivir, Dexamethasone, low molecular weight heparin) in combination with an antiplatelet agent in low doses (100 mg of Acetylsalicylic acid). Upper abdomen CT scan was conducted in order to rule out the case of functional asplenia or an active malignancy. Both patients had a quick and significant clinical improvement, their need for O_2 supply gradually decreased and so they were both discharged from the hospital 8 days after their admission (10 days after the beginning of symptoms).

Follow-up laboratory tests 19 days after the beginning of symptoms and 10 days after the patients' discharge showed a rapid decrease in platelet count in both cases. These findings make the diagnosis of secondary thrombocytosis due to inflammatory response in SARS-COV2 infection the most likely diagnosis. The main laboratory findings are outlined in the chart below (Table 1).

Table 1. Main laboratory parameters.

Laboratory Parameters	Day 0	Day 6	Day 11
Patient A			
WBC ($10^3/\mu L$)	8	9.1	6
HGb (g/dL)	11.5	11.5	11.8
PLT ($10^3/\mu L$)	609	697	343
D-Dimers (mg/l)	0.87	-	-
C.R.P (mg/l)	61.2	6.44	-
Patient B			
WBC ($10^3/\mu L$)	6.4	7	5.8
HGb (g/dL)	9.9	9.4	10.5
PLT ($10^3/\mu L$)	901	753	188
D-Dimers (mg/l)	1.46	-	-
C.R.P (mg/l)	53.5	6.94	-

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Discussion

Secondary thrombocytosis is a common finding with a variety of stimuli including systemic infections, inflammatory conditions, malignancies, acute bleeding and iron deficiency anemia. However, thrombocytosis is a rare manifestation in COVID-19 as the disease is mainly associated with mild thrombocytopenia [1-3]. In a retrospective study only 1.1% of subjects had a platelets count ≥ 600×10^9/L. In this study, patients with thrombocytosis showed significantly better overall survival in comparison with normal and low platelet patients [5]. Thrombocytosis was related with higher VTE incidence but not with higher risk for arterial thrombosis and patients with thrombocytosis had a higher risk of being diagnosed with active malignancy and, especially, gynecological cancer [5]. The blood relation of our patients poses the question of a potential genetic predisposition for the development of secondary thrombocytosis due to SARS-COV2 infection.

Conclusion

The proper treatment and surveillance of this subgroup of COVID-19 patients is unclear. The addition of an antiplatelet agent to the standard of care treatment of COVID-19 seems to be a reasonable choice [6]. As it was previously mentioned, the decrease in platelet count could be associated with lung injury, immune system activation and platelet consumption due to microthrombosis [3]. We could, therefore, suggest that high platelet count is an indirect sign of the absence of those aggravating factors explaining the better overall survival in those patients. Nevertheless, additional evidence is necessary.

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