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A Commentary on Evaluation of Platelet Parameters in Patients with Secondary Failure of Platelet Recovery and Cytomegalovirus Infection after Hematopoietic Stem Cell Transplantation

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Abstract

The number of platelets in SFPR patients decreased significantly, however, in the study by Zhao, et al., Platelet-Large Cell Ratio (P-LCR), Mean Platelet Volume (MPV), platelet distribution width (PDW) of the SFPR patients increased significantly, indicate increased platelet volume and platelet activation, which broadened the scope of SFPR research. But the specific indicators of platelet activation were not measured and the molecular mechanism of platelet activation is unclear in this article. In the future, it is necessary to detect the specific indicators of platelet activation (such as P-Selectin and TXB2) and explore the molecular mechanism of platelet activation in SFPR patients. In addition, CMV infection is a risk factor for the development of SFPR. In the study by Zhao, et al., the authors discovered for the first time that mild CMV infection directly activated platelets, which were inhibited in the presence of severe CMV infection. This provides theoretical guidance for the clinical treatment of SFPR patients with CMV infection.

Keywords: Secondary failure of platelet recovery; Cytomegalovirus; Infection; Platelet Parameters

Description

Hematopoietic stem cell transplantation provides an effective choice to treat a variety of malignant and non-malignant diseases [1]. Secondary Failure of Platelet Recovery (SFPR) is one of the common complications after hematopoietic stem cell transplantation, and fatal bleeding is prone to occur [2]. Relevant studies mainly involve risk factors and treatment of SFPR; no studies were reported about platelet activity in SFPR patients [2]. In addition, cytomegalovirus infection is also a common complication after hematopoietic stem cell transplantation [3]. Studies have shown that cytomegalovirus infection can activate platelet but it is unclear whether Cytomegalovirus (CMV) infection in SFPR patients affects platelet activity [4]. Recently, a study by Zhao, et al., highlighted altered platelet activation in patients with SFPR and the impact of different loads of CMV infection on platelet activity in SFPR patients after hematopoietic stem cell transplantation [5].

Platelet activation is an important process for platelet function. Correct assessment of platelet activity in disease states is critical for patients [6]. Clinically, Mean Platelet Volume (MPV), Platelet Distribution Width (PDW), and Platelet-Large Cell Ratio (P-LCR) are commonly used to evaluate platelet activation in patients. In Primary Open-Angle Glaucoma, the combined use of MPV, PDW, and P-LCR has been reported to evaluate platelet activation [7]. In the study by Zhao, et al., the authors compared the platelet-related parameters of healthy donors, non-SFPR patients and SFPR patients and found that MPV, PDW and P-LCR of SFPR patients were significantly higher than those of healthy donors and non-SFPR patients. This suggests increased platelet activation in SFPR patients. That suggest that there may not be a need to rush administering platelet transfusion after thrombocytopenia in patients with SFPR and that enhanced platelet activity might partially reduce the risk of bleeding caused by thrombocytopenia. However, the author did not measure the specific indicators of platelet activation, which is regrettable. Future studies may need detect of P-selectin and GP IIbIIIa complex, etc. to support the conclusions of author. In addition, the molecular mechanism associated with increased platelet activation in SFPR patients needs to be elucidated in the future.

CMV infection is the one of common infectious complication after Hematopoietic Stem Cell Transplantation (HSCT) [3]. Studies have reported that in patients with atherosclerosis, CMV can infect and activate platelet and promote the formation of neutrophil-platelet complexes, thereby accelerating the progression of atherosclerosis [4]. Another study showed that the higher the CMV-DNA copy number, the lower the viral clearance rate after treatment, and compared with

the patients with low CMV copy number, the patients with high CMV copy number had a higher recurrence rate after treatment [8]. Therefore, Zhao, et al., [2] divided the patients after hematopoietic stem cell transplantation into three groups according to the CMV load (CMV negative group, <8000 CMV DNA copies/mL group and >8000 CMV DNA copies/mL group), and compared the platelet parameters among the three groups. That found for the first time that mild CMV infection can increase platelet activity in patients after HSCT. This suggests that we do not need to rush to eliminate the virus in the human body after CMV infection in patients after HSCT. Drugs can be used to control the virus at a low copy level, so that the platelets of patients after HSCT can be maintained in a state of increased activation and reduce the risk of bleeding. According to the CMV load, SFPR patients were divided into three groups (CMV negative group, <8000 CMV DNA copies/mL group and >8000 CMV DNA copies/mL group) and the platelet parameters of the three groups were compared. It was also found for the first time that severe CMV infection could inhibit platelet activation in SFPR patients, which suggested that SFPR patients should be treated with antiviral therapy immediately after severe CMV infection to avoid fatal bleeding. However, the molecular mechanism of CMV on the bidirectional regulation of platelet activity in post-transplantation patients has not been elucidated and needs to be studied in the future.

In conclusion, zhao, et al., [5] in their recent publication concluded that increased platelet activation in SFPR patients and inhibition of platelet activation in SFPR patients with severe CMV infection. This is of great significance for the clinical treatment of SFPR patients.

Authors Contribution

Both authors have conceptualized, wrote this Short Commentary, reviewed and approved for publication.

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