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721.ALLOGENEIC TRANSPLANTATION: CONDITIONING REGIMENS, ENGRAFTMENT AND ACUTE TOXICITIES

Treatment with Hetrombopag for Poor Platelet Engraftment after Allo-HSCT—a Single Center Retrospective, Observational Study

Gangping Li¹, Yongqi Wang², Ziye Li², Fangfang Yuan, MD², Minghui Li², Yuewen Fu²¹ the Affiliated Tumor Hospital of Zhengzhou University, Henan Provincial Cancer Hospital, Zhengzhou, China² the Affiliated Tumor Hospital of Zhengzhou University, Henan Provincial Cancer Hospital, Zhengzhou, China

Background: Allogeneic hematopoietic stem cell transplantation (allo-HSCT) is a common and important treatment for most hematopoietic diseases. Some patients may experience poor platelet engraftment after allo-HSCT, which may lead to transplant failure and is also an important influencing factor for post-HSCT complications such as graft versus host disease (GVHD) and non-relapse mortality (NRM). Thus, exploring methods to improve platelet engraftment is very important. Hetrombopag is a small-molecule thrombopoietin-receptor agonist (TPO-RA) produced in China, stimulates the proliferation and differentiation of megakaryocytes and promotes platelet production through binding to thrombopoietin receptor to stimulate multiple intracellular signaling pathways. It has been reported that hetrombopag was effective in promoting platelet engraftment and decreasing platelet transfusion after allo-HSCT. Here, we studied the efficacy and safety of hetrombopag for poor platelet engraftment after allo-HSCT, of whom did not respond to rhTPO treatment.

Aims: To evaluate the efficacy of hetrombopag in patients with poor platelet engraftment after allogeneic hematopoietic stem cell transplantation.

Methods: In this retrospective study, data from patients with poor platelet engraftment after allo-HSCT who had been treated firstly with rhTPO and then with hetrombopag at Henan Cancer Hospital from July 1, 2021 to July 1, 2023 were collected. Patients should meet the following criteria to enroll in the study: experienced poor platelet engraftment who had been treated with rhTPO from day +4 post-HSCT. Here we defined poor platelet engraftment as platelet count (PLT) < 20 × 10⁹/L beyond day +28 post-HSCT. We analyzed 57 patients mentioned above and divided them into two groups in a ratio of 1:2, 19 of them were given hetrombopag until their PLT exceeded 50 × 10⁹/L and maintained for more than 7 days without platelet transfusion (hetrombopag group), another 38 patients were treated without hetrombopag (control group). The initial dose of hetrombopag was 5, or 7.5 mg/d according to the patient's conditions and characteristics (e.g., age, PLT, and bleeding risk). After allo-HSCT, patients administered rhTPO at a dose of 300U/kg·d from day +4. We defined rhTPO as ineffective when PLT was still below 20×10⁹/L beyond +21 days post-HSCT after rhTPO administration. At this time, rhTPO was replaced with hetrombopag, or IL-11, decitabine, platelet transfusion, etc.

Results: The median ages of the two groups of patients were 38 years (hetrombopag group) and 41 years (control group), respectively. The median baseline platelet count in both groups were 20 × 10⁹/L. The proportion of patients with PLT recovery to 50×10⁹/L was significantly higher in the hetrombopag group than in the control group (47.3% vs 7.9%, $\chi^2=26.03$, $P<0.0001$). The median time to PLT recovery to 50×10⁹/L was 10 (7-48) days in the hetrombopag group and 33 (21-94) days in the control group ($P=0.004$). The mean units of platelet transfusion in the hetrombopag group was significantly lower than that in the control group (2.7 vs 6.3 U, $P=0.001$), and no thrombotic events occurred. There was no statistical difference in the incidence of aGVHD (42.0% vs 44.7%, $P=0.850$) and NRM (5.2% vs 7.9%, $P=0.714$) between the two groups, suggesting that hetrombopag had no significant effect on aGVHD and NRM.

Conclusion: For allo-HSCT patients who experienced poor platelet engraftment after rhTPO treatment, hetrombopag can rapidly promote PLT recovery, reduce the units of platelet transfusion, and have no risk of the increase of aGVHD and NRM. These results still need further validation in a larger sample prospective confirmatory study in the future.

Disclosures No relevant conflicts of interest to declare.

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