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Opinion Article

Hematopoietic Stem Cell Transplantation and Acute Kidney Damage

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DESCRIPTION

Aplastic anaemia and thalassemia are examples of bone marrow failure syndromes and lymphoproliferative illnesses that can be effectively treated with Hematopoietic Stem Cell Transplantation (HSCT). One of the most widely used treatments for the treatment of neoplastic illnesses, especially lymphoproliferative disorders and states of bone marrow failure, such as aplastic anaemia and thalassemia's, is Hematopoietic Stem Cell Transplantation (HSCT) many transplants are performed annually all throughout the world. Nevertheless, the increasing number of adverse effects that have been reported following the operation has restricted the usage of HSCT. This study discusses about acute kidney damage, one of the main side effects of HSCT (AKI).

There have been a number of studies comparing the prevalence of AKI among various HSCT methods. The majority of HSCT patients have AKI within a year of their procedure. Unfortunately, the way that AKI has been defined varies a lot between researches. The precise incidence of AKI is therefore difficult to calculate. Nowadays, reports on the prevalence of AKI range from 20% to 73%. Nonetheless, some research has revealed numbers as high as 92. The usage of several HSCT kinds is presently widespread. They include autologous HSCT, nonmyeloablative allogeneic therapy, and myeloablative allogeneic therapy.

Prior to the infusion of donor cells that are matched for the human leucocyte antigen, myeloablative allogeneic HSCT uses a conditioning regimen that consists of chemotherapy and radiation. In myeloablative HSCT, cyclophosphamide, busulfan, cytarabine, and whole body irradiation are employed as conditioning regimens.

Depending on the kind of cancer, different conditioning regimens have been documented in various studies. Prior to the infusion of the donor cells, nonmyeloablative allogeneic HSCT uses a decreased intensity conditioning regimen. Patients with serious comorbidities are better suited for this HSCT method. Fludarabine, busulfan, and cyclosporine are among the conditioning regimens used in nonmyeloablative HSCT. Before to chemotherapy and radiation treatment, the patient's own stem cells are removed for autologous HSCT, and following processing, the same stem cells are infused back into the patient. All recipients of allogeneic transplants are given prophylaxis for Graft Versus Host Disease (GVHD). Cyclosporine A (CsA), Mycophenolate Mofetil (MMF), Tacrolimus (FK), and even short-term Methotrexate (MTX) are used in prophylactic regimens.

Nevertheless, only allogeneic transplants can employ the aforementioned regimens as prophylaxis against GVHD. Also, the majority of the patients described in various research received acyclovir and azoles as prophylaxis against infections. AKI following HSCT has been linked to a higher risk of death and a worse overall survival rate.

The effectiveness of different treatment strategies for preventing this problem has been examined in several trials. N-acetyl cysteine was not shown to be helpful in preventing AKI in myeloablative allogeneic transplant patients. Presently, the cornerstone of management is the avoidance of risk factors connected to the emergence of AKI. Serum creatinine is one of the oldest indicators that has been utilised to determine the criteria of AKI. Although serum creatinine is still the gold standard, alternative biomarkers in this section that have recently demonstrated sensitivity for kidney damage identification, particularly following HSCT. They could serve as guidelines for more actively anticipating and treating AKI in the future.

CONCLUSION

AKI is one of the main drawbacks of HSCT in the treatment of bone marrow failure and lymphoproliferative diseases. The GVHD, usage of nephrotoxic drugs, sepsis, hepatic SOS, tumour lysis syndrome, TMA, and marrow infusion toxicity have all been recognised as risk factors. The avoidance of risk factors that lead to the development of AKI is the main goal of AKI treatment in HSCT patients. If feasible, nephrotoxic drugs should be avoided. Reduced intensity conditioning should also be used. Sepsis, tumour lysis syndrome, hepatic SOS, and marrow infusion toxicity should also be identified and treated promptly. Yet for efficient therapy of AKI and associated consequences, early nephrologist participation in the course of the disease is crucial.

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