characteristics were recorded. GT was administered to patients with an absolute neutrophil count (ANC) $<0.5 \times 10^3/\mu L$ for at least three days, evidence of bacterial and/or fungal infection, and no response to appropriate antimicrobials for at least 48 hours. **Results:** The median age was 42 years (min-max, 19-66 years). The majority of patients were diagnosed with acute myeloid leukemia (AML) (50%)(11/22). The median CRP value was 168.5 mg/dl (min-max, 31.1-360 mg/dl). In 40.9 % of patients who received GT, their primary disease was in complete remission, while in 59.1 %, their primary disease was relapse. The infection etiologies included pneumonia (n=5), sepsis (n=2), pneumonia and sepsis (n=11), pneumonia + sepsis + catheter-associated infection (n=4), catheter-associated infection + mucositis (n=1), and abscess (n=1). Each patient received a median of 3 GTs (min-max, 1-6). The median transfused granulocyte dose per transfusion was 3.5×10^{10} (min-max, 0.8-9.4 $\times 10^{10}$). The median dose transfused, calculated based on the recipient's body weight, was 5.1×10^8/kg (min-max, 0.8-17 $\times 10^8$/kg). On average, the median number of granulocytes transfused per patient was 5.3×10^8/kg (min-max, 1.9-11.3 $\times 10^8$/kg). The median time from HSCT to the first GT was 192 days (min-max, 50-795 days). The median duration of fever before GT was three days (min-max, 2-6 days), and the time until the fever defervescence was 2 days (min-max, 1-5 days). The median duration of neutropenia before GT is 25 days (min-max, 8-30 days). After GTX treatment, A favorable response was observed in 16 of 24 infection episodes (66.7%) regarding the resolution of infections. In 4 of the 8 infection episodes where the infection did not resolve, the patient also had a relapse of the disease. In 5 of 12 infection episodes that required intensive care, the need for intensive care was eliminated after GT. A statistically significant difference was found between the time of GT initiation and the ANC, TLC, and PLT counts on the fourth-day post-GT (p =0.001, p=0.001, p=0.003, separately for ANC, TLC, and PLT). The median follow-up in our cohort of patients is 600 days. The 30-day and 100-day OS were 67.7% and 50%, respectively. A mortality rate by day-28 was 3.8% and mortality rate by 100 was 19.2%. Acute, chronic GVHD, and CMV reactivation were not observed. **Conclusion:** GT therapy may be effective in many critically ill patients with prolonged and profound neutropenia. It may be more beneficial in select patients, as it provides more time to overcome infections resistant to broad-spectrum antibiotics. Larger randomized trials are needed to confirm the effectiveness of GT in such patients.

https://doi.org/10.1016/j.htct.2025.106130

OP 21

CATATONIA FOLLOWING IFOSFAMIDE CHEMOTHERAPY IN A PATIENT WITH HISTIOCYTIC SARCOMA: A RARE NEUROPSYCHIATRIC COMPLICATION

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Introduction: Histiocytic sarcoma (HS) is a rare, aggressive malignancy of monocyte–macrophage lineage, typically presenting with extranodal disease and lacking B- or T-cell markers [1]. Because of its rarity, there is no standard treatment, though salvage regimens such as ICE (ifosfamide, carboplatin, etoposide) have demonstrated some benefit. Ifosfamide, a DNA-alkylating prodrug metabolized by hepatic CYP3A4 and CYP2B6, is associated with central nervous system (CNS) toxicity in 10–30% of patients [2,3]. Encephalopathy is the most common presentation, while catatonia—characterized by stupor, mutism, negativism, posturing, and waxy flexibility—is rarely reported in oncology patients [4]. **Case Presentation:** A 27-year-old male with stage IV HS, confirmed by biopsy of an 80 × 70 mm terminal ileum mass, was admitted for ICE chemotherapy. On day three, he developed acute psychomotor symptoms including stupor, mutism, and negativism. The Bush–Francis Catatonia Rating Scale (score 7) and Kanner Catatonia Screening Instrument (score 4) confirmed retarded-type catatonia. Neurological evaluation (cranial CT, diffusion-weighted MRI) and laboratory studies were unremarkable. Vital signs remained stable. He was treated with intravenous diazepam 10 mg every 8 hours (two doses total), leading to full resolution of catatonic symptoms. The patient was discharged clinically stable. **Conclusion:** Discussion Ifosfamide-induced neurotoxicity typically appears within 48 –72 hours, mediated by toxic metabolites such as chloroacetaldehyde that disrupt mitochondrial function and neurotransmission [2,3]. While encephalopathy is well-documented, catatonia is extremely rare and underrecognized. In this case, the temporal relationship to ifosfamide, absence of structural CNS pathology, and rapid benzodiazepine response strongly support ifosfamide-induced catatonia. Similar observations have been described rarely; Gupta et al. [5] reported an analogous case in lymphoma. Benzodiazepines remain first-line therapy, often producing rapid resolution, even in drug-induced catatonia [6].Conclusion This case highlights catatonia as a rare neuropsychiatric complication of ifosfamide. Recognition of such unusual adverse effects is critical, as early diagnosis and benzodiazepine treatment can prevent delays in cancer therapy and improve outcomes.

https://doi.org/10.1016/j.htct.2025.106131

Stem Cell Transplantation

OP 22

RESULTS OF AUTOLOGOUS HEMATOPOIETIC STEM CELL TRANSPLANTATION IN REFRACTORY MULTIPLE SCLEROSIS: TWO CASE REPORTS

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