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Case study: Acute plasmoblastic leukemia presentation following effective haploidentical hematopoietic stem cell transplantation therapy

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Abstract

Plasma cell leukemia is a rare and aggressive malignancy characterized by monoclonal gammopathy and the presence of circulating plasma cells in the peripheral blood. Therapeutic strategies for plasma cell leukemia remain undefined, with treatments primarily borrowed from those used in multiple myeloma. The first-line treatment typically involves a combination of a proteasome inhibitor, an immunomodulatory agent, steroids, and/or anthracyclines and alkylators within an intensive chemotherapy regimen. Following this, consolidation with autologous hematopoietic stem cell transplantation is offered to eligible patients, followed by maintenance therapy. For patients ineligible for autologous hematopoietic stem cell transplantation, allogeneic hematopoietic stem cell transplantation is considered a viable alternative. Given the challenges in securing a fully human leukocyte antigen–matched donor, haploidentical hematopoietic stem cell transplantation serves as a potential salvage therapy, as demonstrated in the clinical case presented. This article presents the case of a female patient in her 50s diagnosed with plasma cell leukemia who, following unsuccessful autologous hematopoietic stem cell mobilization, underwent haploidentical hematopoietic stem cell transplantation from her son, resulting in complete donor chimerism and a favorable response.

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Introduction

Plasma cell leukemia (PCL) is a rare and challenging plasma cell dyscrasia characterized by an unfavorable prognosis. The taxonomy of PCL includes primary PCL (pPCL) and secondary PCL (sPCL), with the latter developing after a prior diagnosis of multiple myeloma (MM). pPCL is frequently observed, constituting 60%–70% of all PCL cases, whereas the incidence of sPCL ranges from 30% to 40%.[1] This secondary manifestation typically arises in the advanced stages of MM and is diagnosed in approximately 0.5%–4.0% of all patients with MM.[2]

In adherence to the diagnostic standards set forth by the International Myeloma Working Group (IMWG) and the World Health Organization (WHO), PCL diagnosis is validated by meeting either of the two criteria. Specifically, the presence of $\geq$20% circulating plasma cells in the peripheral blood or an absolute plasma cell count surpassing 2×10^9/L is considered sufficient for the conclusive diagnosis of PCL.[3]

The clinical presentation of PCL differs significantly from that of MM in several aspects, as reflected in Table 1.

Key laboratory abnormalities in PCL include hypercalcemia, defined as a total (unionized) calcium level of >2.75 mmol/L or an ionized calcium level of >1.45 mmol/L.[4–17]

Typically, pPCL manifests in a demographic population skewed toward younger patients, predominantly during the advanced phases of the disease, often accompanied with extramedullary lesions such as hepatomegaly, splenomegaly, and involvement of other organs. Conversely, osteolysis is less frequently observed.[18] Given the heightened prevalence of extramedullary lesions, the IMWG has advocated the inclusion of 18 F-fluorodeoxyglucose positron emission tomography (FDG-PET/CT) as a crucial diagnostic, evaluative, and monitoring tool for PCL. This recommendation underscores the importance of advanced imaging modalities in comprehensively characterizing the disease presentation, progression, and response to therapeutic interventions.

Table 1. Clinical characteristics and cytogenetics in primary plasma cell leukemia and multiple myeloma.

Clinical characterization and cytogenetics	PCL	MM
Nonsecretory	10%	4%
Other	1%	1%
Anemia, Hgb < 100 g/dL	81%	47%
Platelets < 130/L	63%	5%
Elevated creatinine	22%	24%
Abnormal LDH	60%	12%
Hypercalcemia	27%	12%
Bone disease	65%	77%
ISS I	10%	27%
ISS II	23%	39%
ISS III	67%	34%
Translocation (11;14)	26%	21%
Translocation (4;14)	14%	14%
Translocation (14;16)	20%	4%
Deletion (17p)	40%	11%
Full/partial deletion of 13q	42%	48%
Amplification 1q	32%	40%

Hgb: hemoglobin; LDH: lactate dehydrogenase; ISS: international staging system.

Owing to the aggressive trajectory of PCL, the imperative for prompt treatment initiation has been underscored. Typically, patients in this cohort demonstrate a prompt response; however, the aggressive nature of the disease leads to early relapses, a characteristic hallmark posing challenges in the therapeutic landscape of PCL.[5–9,19–21] Conventional cytostatic chemotherapy has yielded unsatisfactory outcomes in the treatment of PCL. Subsequent to the advent of hematopoietic stem cell transplantation (HSCT), coupled with the integration of the proteasome inhibitor bortezomib and immunoregulatory drugs such as thalidomide and lenalidomide, a certain degree of prognosis enhancement has been achieved. Nevertheless, the outcomes still fall short of the optimal therapeutic objectives. This underscores the ongoing imperative for refining treatment strategies in the relentless pursuit of improved outcomes for individuals afflicted with PCL. The reporting of this study conforms to the Case Report (CARE) guidelines.[22]

Case presentation

A female patient in her 50s was diagnosed with PCL in 2022, following the onset of pain in the lumbosacral region with irradiation to the left lower limb, limited mobility, weakness, and numbness in the left lower limb. In March 2022, she sought medical help at her place of residence, where the diagnosis was verified as "PCL, FLC kappa." Examination revealed the presence of free kappa light chains (kappa LCC), free kappa chains (3.71 μg/mL) (C), free lambda chains (0.46 μg/mL) (C); kappa/lambda index of 8.1275; and presence of plasma cells on evaluation of a normal peripheral blood smear (48%), with the characteristic immunophenotype CD138+/CD38-/Kappacyt+/Lambdacyt-/CD117-/CD56+/CD19-/CD20-/is (72.0%). Furthermore, it revealed the presence of ≥10% monoclonal plasma cells in the bone marrow with the characteristic immunophenotype CD138+/CD38-/Kappacyt+/Lambdacyt-/CD117-/CD56+/CD19-/CD20+/is (61.2%). The patient had splenomegaly, serum β2-microglobulin level of 12.8 mg/L, and albumin level of 36.1 g/L; moreover, immunofixation of blood and urine did not reveal any findings. Based on these indications, the patient was diagnosed with stage III according to the international staging system.

Following the confirmation of diagnosis, two cycles of chemotherapy, adhering to the "VDT-PACE" regimen, were administered. Subsequent reassessment revealed a "complete objective response" in accordance with the criteria outlined by the IMWG. General blood analysis: leukocytes, 2.2/L; erythrocytes, 3.52/L; hemoglobin, 114 g/L; and platelets, 149/L. Myelogram: plasma cells, 1%. Conclusion: The presented preparations from the bone marrow punctate were hypocellular. Differential count of myelocaryocytes was performed in 200 cells. Immunophenotyping of peripheral blood. Conclusion: In the examined peripheral blood sample, the population of cells exhibited the immunophenotype CD138+/CD38-/Kappacyt+/Lambdacyt-/CD117-/CD56+/CD19-/CD20-/is (0.0%). Immunofixation of blood serum and urine were negative. Given this favorable response, the decision was made to proceed with autologous HSCT (auto-HSCT).

In November 2022, mobilization of peripheral hematopoietic stem cells (HSCs) was initiated using the etoposide and colony-stimulating factor regimen. The initial CD34+ count on day +11 was insufficient for collection, prompting continued stimulation. The subsequent counts on days +12, +13, and +14 demonstrated incremental improvement, culminating in a count of 3.13% ± 0.06%. Despite these efforts, mobilization failure was confirmed.

Mobilization by the CXCR4 chemokine receptor antagonist plerixafor was not possible due to the unavailability of this drug in Kazakhstan. This drug is purchased by patients themselves, but not everyone has the opportunity to purchase it given the cost, which limits its use.

In response to this, the therapeutic approach shifted to maintenance therapy with lenalidomide from August 2022 to May 2023. Considering the disease status marked by a complete response, unsuccessful HSC mobilization, aggressive disease course, and elevated risk of relapse, alongside the presence of a partially compatible donor, the inclusion of haploidentical T-cell replete graft stem cell transplantation (haplo-TGSCC) from the son was contemplated as the subsequent step in the treatment plan (Table 2).

In February 2023, a retrospective study conducted by the Center for International Studies revealed a 3-year overall survival (OS) of 39% for the specific diagnosis under consideration with allogeneic HSCT (allo-HSCT).[23]

Subsequently, in May 2023, the patient was admitted to the Bone Marrow Transplantation department of the National Research Oncology Center in Astana for haplo-HSCT from her son. The transplantation procedure involved high-dose chemotherapy (HDCT), specifically using fludarabine (30 mg/m^2) and melphalan (MEL; 140 mg/m^2). Haplo-TGSCC from her son was performed, with a CD34 count of 7.7 million/kg. Cyclophosphamide was administered on +3 and +4 days at a dose of 50 mg/kg/day, followed by tacrolimus initiation from +24 days onward at a dose of 0.03 mg/kg/day to mitigate graft-versus-host reaction (GVHR). Subsequent adjustments to tacrolimus dosage were made based on blood concentration levels. Neutrophilic engraftment occurred by +17 days. After achieving complete donor chimerism at +30 days (99.9%), a control bone marrow puncture and donor chimerism assay were conducted at +60 days post haplo-TGSCC. The results indicated an absence of detectable plasma cells in the bone marrow, with the preservation of complete donor chimerism at 98.67%. Immunosuppressive therapy (IST) with tacrolimus was continued until day +195; however, given the absence of GVHR and the status of the underlying disease, IST was discontinued.

Medical history

A female patient in her 50s was admitted to the Center for Hematology Oncohematology and Bone Marrow Transplantation with a confirmed diagnosis of PCL, prompting the consideration of haplo-HSCT. This procedure involved utilizing stem cells from her son, who had achieved a "complete response" following two courses of chemotherapy (HT) using the "VDT-PACE" regimen (bortezomib, dexamethasone, thalidomide, cisplatin, adriamycin, cyclophosphamide, and etoposide). The response was assessed based on the criteria established by the IMWG.[24]

Table 2. HLA genotypes of the recipient and donor for haploidentical stem cell transplantation.

HLA genotype of the recipient				
A*32:01;33:01	B*18:01;56:01	C*01:02;07:01	DRB1*13:01	DQB1*06:03
HLA genotype of the donor				
A*01:01;33:01	B*08:01;56:01	C*01:02;07:01	DRB1*12:01;13:01	DQB1*03:01;06:03

HLA: human leukocyte antigen.

The decision to perform haplo-HSCT was influenced by the patient's prior unsuccessful mobilization of peripheral HSCs, rendering auto-HSCT unfeasible. Faced with the unavailability of a fully human leukocyte antigen (HLA)–matched donor, the patient underwent successful haplo-HSCT from her son, marking a milestone at 10 months post-diagnosis during the initial remission phase. Signed consent for treatment as well as for publication of the study results was obtained from the patient.

Discussion

PCL represents a rare malignancy characterized by an aggressive clinical course and unfavorable prognosis. The scarcity of comprehensive therapeutic outcome data, coupled with a dearth of randomized studies, complicates the decision-making process for treating this disease.

This case report illuminates a successful outcome in a patient who underwent haploidentical T-cell replete graft stem cell transplantation (haplo-TGSCC) from her son. In the dynamic landscape of modern medicine, continuous evolution brings forth new diagnostic techniques and therapeutic approaches, necessitating a nuanced consideration of disease-related, patient-related, and prior therapy-related factors to tailor an optimal treatment strategy. Disease-related factors encompass the quality and duration of response to previous therapies as well as the aggressiveness of relapse. Concurrently, patient-related considerations encompass pre-existing toxic effects, comorbidities, quality of life, age, and performance status.

A notable advancement in recent times is the success achieved with haploidentical transplantation. With the advent of improved GVHR prophylaxis, this approach has become accessible for nearly all eligible patients and has demonstrated efficacy comparable to allo-HSCT. Haplodonors, in this context, share one of their two HLA-A, -B, -C, -DRB1, -DQB1, -DPB1 haplotypes with the patient through genetic inheritance, whereas the other haplotype remains distinct. This breakthrough in haploidentical transplantation presents a promising avenue in the intricate landscape of PCL treatment.

Consequently, haploidentical donors typically exhibit mismatches with the patient in six of the 12 HLA alleles, although the specific number may exhibit variability. Genetic recombination, which occurs at a frequency of up to 1% if the patient and donor lack a direct familial relationship,[20] introduces the potential for mismatches even on the shared haplotype. In the case of our patient, the haplodonor candidate is a son, obviating the necessity to account for recombination. Notably, however, the consideration of excluding recombination is generally advised for all haplodonor candidates, except for those who are the mother, father, son, or daughter of the patient.

The Center for International Blood and Marrow Transplant Research (CIBMTR) study, published in May 2012, reported on outcomes comparing the results of a total of 147 patients, including 97 patients with pPCL who underwent auto-HSCT and 50 patients with pPCL who underwent allo-HSCT within 18 months of diagnosis between 1995 and 2006, the majority of whom (68%) received a myeloablative conditioning regimen.[25–26] The median ages of the two groups were 56 and 48 years. Progression-free survival (PFS) at 3 years was 34% (95% CI, 23%–46%) in the autologous group and 20% (95% CI, 10%–34%) in the allogeneic group. The cumulative recurrence rate after 3 years was 61% (95% CI, 48%–72%) in the autologous group and 38% (95% CI, 25%–53%) in the allogeneic group. The OS after 3 years was 64% (95% CI, 52%–75%) in the autologous group and 39% (95% CI, 26%–54%)

in the allogeneic group. Recurrence-free mortality (RFM) after 3 years was 5% (95% CI, 1%–11%) in the autologous group and 41% (95% CI, 28%–56%) in the allogeneic group. The lack of a positive effect on OS in allo-TGSCC may be partly due to a higher treatment-related mortality (41% in allo-TGSCC versus 5% in autoTGSCC). This study demonstrated that although allo patients had a significantly lower recurrence rate, their RFM was significantly higher with no improvement in OS after 3 years.[25]

In 2020, CIBMTR reported a further analysis of 348 patients with pPCL transplantation between 2008 and 2015. There was an increase in hematopoietic cell transplant utilization from 12% in 1995 to 46% in 2009; however, the outcomes remained poor, with no increase in OS in the allo group compared with that in the previous study.[27]

Initial comparisons were also made between allo-first and first autograft patients (regardless of subsequent second graft insertion).

- For the first auto-HSCT, the estimated 3-year PFS was 14.3 months, with an OS of 33.5 months.
- For the first allo-HSCT, the estimated 3-year PFS was 11.7 months, with an OS was of 17.5 months.

After 60 months, the probabilities of OS and PFS were similar: OS: allo (34.6%), auto (31.3%); PFS: allo (19.9%), auto (14.3%). This study confirms that significant PFS occurs in patients undergoing allo-HSCT as a first transplant.

In June 2022, the European Society for Blood and Marrow Transplantation published an article presenting a comparative analysis of auto- and allo-HSCT strategies in patients with pPCL, employing dynamic prediction modeling.[26] Through retrospective analyses across distinct time periods, the study substantiates a significant risk of mortality within the initial 100 days for allo-primary transplantation, advocating the preference for tandem transplantation strategies. However, it emphasizes that the disease status, specifically the remission status, and the attainment of complete response before the initial transplant plays a pivotal role in determining the optimal form of treatment for patients with pPCL.

Despite advancements attributed to the integration of novel agents, pPCL persists as a formidable challenge for clinicians. This retrospective study contributes valuable evidence to guide transplant clinicians in their decision-making processes, offering insights to patients regarding the most appropriate approach tailored to their circumstances following effective induction therapy. The discernments derived from this study have the potential to inform and optimize clinical decision-making processes related to transplant strategy for patients suffering from with the complexities of pPCL.

Conclusions

PCL stands out as a distinct entity from MM, both clinically and genetically. Despite this distinction, therapeutic strategies for PCL remain inadequately defined, leading to the adoption of regimens originally developed for MM. The prognosis for PCL is notably poor, with a median OS of <1 year. Nevertheless, advancements have been witnessed with the introduction of HDCT supported by stem cells, along with the inclusion of bortezomib and lenalidomide in treatment protocols.

Our clinical observations underscore a positive treatment outcome associated with the adoption of haplo-HSCT as a salvage option. In our specific case, the patient

underwent HDCT complemented by donor stem cell support, revealing promising results in the context of PCL management. These findings contribute to the growing body of evidence supporting the efficacy of haplo-HSCT as a viable therapeutic avenue in the intricate landscape of PCL.

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Author contributions

Conceptualization: Aigerim Najipova, Azat Karabekov, and Vadim Kemaykin; methodology: Azat Karabekov and Aigerim Najipova; formal analysis: Madina Abdirgalieva; writing—original draft preparation: Aigerim Najipova, Madina Abdirgalieva, Azat Karabekov, Olga Kolesnikova, Ruzal Vildanova, Almira Manatova, and Zhuldyz Kuanysh; writing—review and editing: Aigerim Najipova, Madina Abdirgalieva, Azat Karabekov, Olga Kolesnikova, Ruzal Vildanova, Almira Manatova, and Zhuldyz Kuanysh; supervision: Azat Karabekov and Vadim Kemaykin; and project administration: Almira Manatova and Zhuldyz Kuanysh. All authors have read and agreed to the published version of the manuscript.

Consent for publication

Informed consent for publication was obtained for all participant data included in this study.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Declaration of conflicting interests

The authors declare no conflict of interest.

Ethical approval statement

The study was conducted in accordance with the Declaration of Helsinki and approved by the Local Ethics Committee of National Research Oncology Center, Kazakhstan (permit number no. 15).

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