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Remission of refractory Sezary syndrome after bone marrow transplantation from a matched unrelated donor

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(Received 5 February 1999; accepted 27 July 1999)

ABSTRACT

Sezary syndrome is a leukemic variant of mycosis fungoides (MF)/cutaneous T-cell lymphoma (CTCL). Bone marrow transplantation (BMT) from a matched unrelated donor was performed in a 22-year-old woman with a 10-year history of Sezary syndrome who had failed treatment with corticosteroids, methotrexate, photochemotherapy, photopheresis, hydroxyurea, interferon-α, and cladarabine. At the time of BMT, she had persistent erythrodermic skin disease, adenopathy, circulating Sezary cells and bone marrow (BM) involvement. The patient underwent BMT from a 6/6 HLA-matched unrelated male donor in August 1996. A BM biopsy obtained on day 30 after BMT showed no evidence of lymphoma and complete male donor engraftment. Her skin lesions resolved within 100 days after transplant. Complete staging studies, including T-cell receptor gene rearrangement studies performed at 36 months post-BMT, showed no evidence of recurrent Sezary syndrome. This represents her first durable remission since the initial diagnosis more than 12 years ago. To our knowledge, this is the first patient with refractory Sezary syndrome who has been successfully treated with allogeneic unrelated donor BMT. Our results indicate that this modality may be effective in inducing remission in refractory MF/CTCL, including Sezary syndrome.

KEY WORDS

Bone marrow transplantation • Cutaneous T-cell lymphoma • Sezary syndrome

INTRODUCTION

Sezary syndrome is a distinct clinical variant of erythrodermic mycosis fungoides (MF) or cutaneous T-cell lymphoma (CTCL) in which patients present with circulating malignant lymphocytes, called Sezary cells, in the peripheral blood [1–4]. In a recent study of 106 patients with erythrodermic MF and Sezary syndrome, patients presenting with >5% circulating Sezary cells had a median survival of 2.6 years [5].

A variety of treatments are currently being used for MF/CTCL, including topically directed therapies such as total skin electron beam radiation therapy, photochemotherapy (PUVA), and topical chemotherapy and systemic therapies such as single-agent chemotherapy, retinoids, combination chemotherapy, interferon-α, extracorporeal photochemotherapy (photopheresis), and combined modality regimens [1–4]. Most of these treatments are palliative and none is considered to have significant curative potential in patients with advanced disease. Currently, photopheresis with or without interferon-α is the standard treatment for erthyrodermic MF and Sezary syndrome [6,7].

Very few patients with MF/CTCL have been treated with high-dose therapy and bone marrow transplantation (BMT). In most of these cases, autologous bone marrow has been used for hematopoietic reconstitution [8,9]. Although feasible, this approach has been associated with a very high risk of relapse. Herein, we report our experience with allogeneic BMT from an unrelated donor in a young woman with refractory Sezary syndrome. To our knowledge, this is the first patient with Sezary syndrome or advanced CTCL who has been successfully treated with allogeneic unrelated donor BMT.

This work was supported in part by U.S. Public Service Grants 30206 and 33572 from the National Cancer Institute. A.M. was supported by a Lymphoma Research Foundation of America Fellowship and an American Cancer Society Clinical Oncology Career Development Award.

CASE REPORT

Clinical history

The patient was 12 years old when she presented in early 1986 with erythodermic follicular mucinosis, leukocytosis, hypereosinophilia, diffuse peripheral adenopathy, and 40% circulating Sezary cells. Immunophenotyping and gene rearrangement studies performed on peripheral blood showed evidence of a T-cell clonal neoplastic process. Initial prednisone therapy was unsuccessful. In July 1986, she developed worsened leukocytosis and infiltrative skin disease, fevers, and progressive pulmonary infiltrates. She received weekly methotrexate but was treated subsequently with photopheresis and PUVA. The initial clinical observations and pathologic and molecular diagnostic studies on this patient were reported in 1988 by LeBoit *et al.* [10].

In 1991, hydroxyurea and prednisone were administered to control her blood counts. In 1994, her white blood cell count became elevated to 54,000/μL in association with marked eosinophilia, lymphocytosis, and persistent Sezary cells. A lymph node biopsy showed morphologic and immunophenotypic findings consistent with a T-cell lymphoma. Additionally, cytogenetic studies on the lymph node tissue revealed clonal karyotypic changes (i.e., trisomy 3 and a derivative chromosome 23) that have been found in some T-cell lymphomas. A bone marrow biopsy and aspirate also showed morphologic evidence of lymphomatous involvement. The patient was started on interferon-α in May 1994 and was maintained on daily doses of 6–9 million units for >2 years, to which she had a partial response. Interferon-α was discontinued 2–3 months before BMT, at which time she received one cycle of cladarabine (2-chlorodeoxyadenosine), with minimal response.

Disease status before BMT

Clinically, the patient had mild to moderate pruritis. On physical examination, there were focal areas of confluent erythema on her abdomen, back, and extremities involving 40–50% of the skin surface area as well as diffuse peripheral adenopathy measuring 2.0 cm in maximum diameter. Computed tomography scans of the chest, abdomen, and pelvis showed bilateral axillary and inguinal adenopathy measuring 2.0 cm.

Peripheral blood showed occasional (<5%) cytologically atypical lymphoid cells consistent with Sezary cells (Fig. 1). A bone marrow biopsy and aspirate showed morphologic evidence of involvement with T-cell lymphoma. Immunohistochemical studies demonstrated cytoplasmic CD3 and CD45RO expression. Flow cytometry was also performed on cells in suspension prepared from the bone marrow aspiration. The neoplastic cells expressed the CD2, CD4, CD5, TCRα/β, and CD45 antigens and were negative for the surface CD3 and CD7 antigens (Fig. 2). Cytogenetics of bone marrow showed a normal XX karyotype.

Details of BMT

Written informed consent for participation in this study was obtained before BMT in accordance with the City of Hope National Medical Center Institutional Review Board guidelines. The patient received a conditioning regimen of fractionated total-body irradiation to a total dose of 1320 cGy followed by cyclophosphamide 60 mg/kg (ideal body weight) for two consecutive days. On 27 August 1996, she underwent BMT using unmanipulated bone marrow from a 6/6 HLA-matched unrelated 35-year-old male donor. Serologic typing was performed for class I HLA-A, -B, and -C loci. Molecular typing of class II HLA-DRB1 and -DQ antigens was done by sequence-specific oligonucleotide probe hybridization [11]. The donor and recipient were serologically matched at HLA-A and -B loci and molecularly matched at HLA-DR loci but mismatched at one HLA-C allele. The patient received 1.0×10^8 mononuclear cells per kilogram of body weight. A $CD34^+$ cell count was not obtained. Graft-vs.-host disease (GVHD) prophylaxis consisted of cyclosporine A (CSA) and prednisone as well as methotrexate on days 1, 3, 6, and 11 [11].

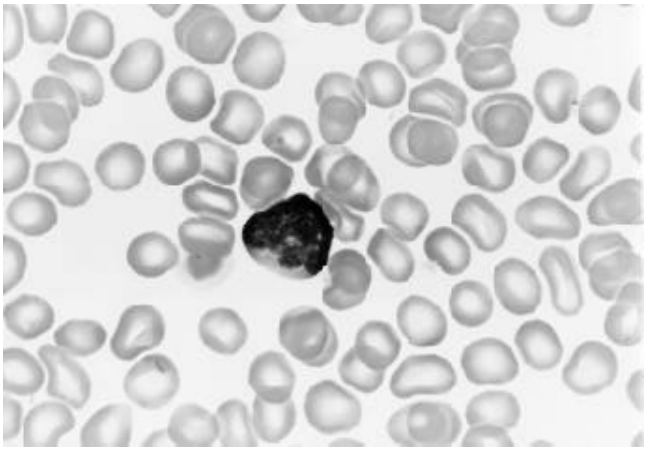

Figure 1.
Occasional slightly larger lymphoid cells (Sezary cells) with irregularly folded, cerebriform nuclei are seen in the patient's peripheral blood (Wright-Giemsa stained smear, original magnification ×1000).

The patient reached an absolute neutrophil count >500/μL on day 16 and platelet count >25,000/μL without subsequent platelet transfusions on day 13. During the period of neutropenia, she developed fevers that responded to broad-spectrum antibiotics. She subsequently recovered from all acute BMT-related toxicities.

Flow cytometry

Immunophenotyping was performed by three-color flow cytometric analysis of bone marrow aspirates with a Becton Dickinson (Mountain View, CA) FACScan instrument [12].

T-cell receptor gene rearrangement studies

The presence of a clonal T-cell receptor gene rearrangement was detected at the time of her initial diagnosis and reported in 1988 by LeBoit *et al*. at the referring institution [10]. These investigators used Southern blot analysis of DNA extracted from peripheral blood involved with 40% Sezary cells to demonstrate the presence of rearranged bands using three different restriction enzymes and a radiolabeled cDNA probe to the TCR-β gene [10]. In the current study of a post-BMT remission specimen, rearrangements of the TCR-γ variable 1–8, 9, 10, and 11 genes were analyzed using the polymerase chain reaction (PCR), as previously described [13].

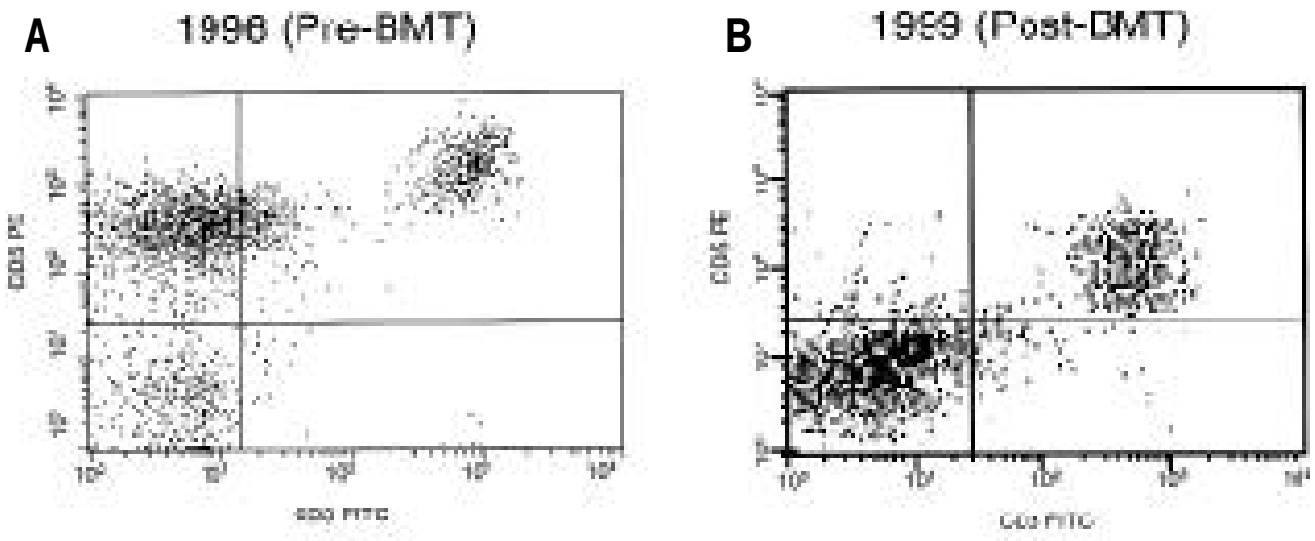

Figure 2.
Flow cytometric immunophenotyping of the 1996 bone marrow specimen involved by lymphoma (A) demonstrates a population of neoplastic T cells that are CD5$^+$ with partial loss of CD3 (upper left quadrant). The follow-up specimen obtained in 1999 (B) demonstrates no abnormal T-cell population.

RESULTS

A bone marrow biopsy and aspirate obtained on day 30 showed complete disappearance of lymphoma. Cytogenetics showed a normal XY karyotype consistent with complete male donor engraftment. The erythema and induration of her skin and symptoms of pruritis resolved within the first 100 days posttransplant. Examination of multiple sequential peripheral blood smears has shown no evidence of circulating Sezary cells during a 36-month follow-up period. She developed dermatomal Herpes zoster before day 100 which was treated with intravenous acyclovir. She had no acute GVHD or cytomegalovirus disease. Her posttransplant course has been complicated by the development of chronic skin GVHD, which is being treated with cyclosporine and prednisone. The flare-ups of GVHD involved primarily sun-exposed areas and did not correlate with previous areas of involvement by CTCL. Staging studies, including computed tomography scans and routine bone marrow biopsies performed at regular post-BMT evaluations, show no evidence of recurrent CTCL.

On physical examination and routine laboratory testing performed 36 months post-BMT, there was no evidence of Sezary syndrome. Her Karnofsky performance status is 80–90%. The patient remains free of pruritis. Flow cytometry analysis of the patient's bone marrow before transplantation revealed a population of CD5$^+$, CD3$^{+/-}$ cells that were no longer present 29 months after transplantation (Fig. 2).

Initially, the clonal nature of the patient's Sezary cells was demonstrated by Southern blot analysis using a generic TCR-β probe (10). Because all T-lymphoid malignancies with TCR-β rearrangements also have TCR-γ rearrangements, which are less heterogenous, we evaluated the patient's posttransplant bone marrow (at 36 months) by PCR using TCR-γ–specific probes (13). No evidence of TCR-γ clonality was found, indicating the prior clonal population was no longer present (Fig. 3).

DISCUSSION

Mycosis fungoides is a rare type of low-grade T-cell lymphoma involving primarily the skin that can present with the formation of patch, plaque, and tumor lesions. Advanced stages of this type of CTCL are associated with dissemination into lymph nodes and visceral sites, features generally associated with a poor prognosis. A variant of this disorder presents with generalized pruritic erythroderma in ~5% of MF/CTCL patients. Frequently, patients with generalized erythroderma develop Sezary syndrome, a leukemic manifestation of this disease characterized by the presence of neoplastic T-cells with hyperlobated cerebriform nuclei (Sezary cells) in the peripheral blood. The prognosis for

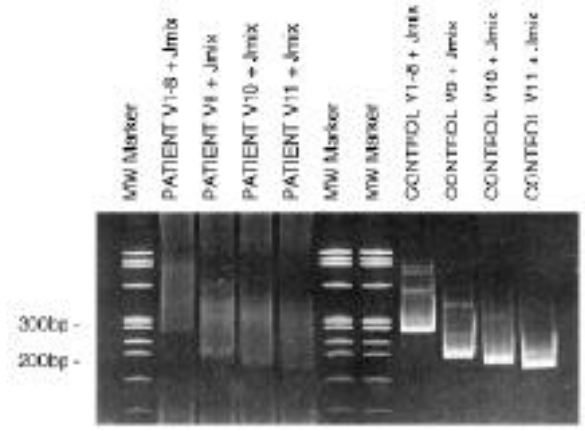

Figure 3.
Polymerase chain reaction study for T-cell receptor γ chain gene rearrangements fail to demonstrate evidence of a monoclonal T-cell population in the 1999 follow-up bone marrow specimen using primer sets designed against all eleven variable (V) regions of the T-cell receptor γ gene. Positive controls for each primer set are illustrated in the last four lanes on the right. MW, molecular weight.

patients with Sezary syndrome is generally poor, with a median survival of <3 years [5].

Most patients with MF/CTCL are in their sixth decade of life. A recent study of prognostic factors in advanced-stage erythrodermic MF and Sezary syndrome reported a mean age of 62 years (range 24–87) in the 106 patients analyzed [5]. There are few reports of MF/CTCL developing in children and adolescents [14–18]. Clinical presentations were primarily patch and plaque stage disease, but rare instances of tumor stage, granulotamous Slack disease, and classic Sezary syndrome have also been reported, suggesting that MF/CTCL in young patients also represents a heterogeneous spectrum of disease. Long-term follow-up results of therapy in this subgroup of patients are not available, but presumably, their prognostic variables are similar to the typical older patients with MF/CTCL. Because these disorders have a marked propensity for relapse and are generally considered to be incurable with conventional treatments, it is likely that younger patients afflicted with MF/CTCL will suffer reduced longevity as a consequence of their malignancy.

The patient reported herein presented with several unique clinical features. In 1986, at the age of 12, she developed follicular mucinosis, a cutaneous disorder which by itself is not considered to be a malignant process, along with immunophenotypic and immunogenetic evidence of clonal neoplastic Sezary cells of T-cell lineage in the peripheral blood [10]. The natural history of her illness was characteristic of Sezary syndrome with the development of peripheral adenopathy, leukocytosis, eosinophilia, erythroderma, alopecia, pulmonary infiltrates, and circulating malignant T-cells. She failed multiple conventional therapies aimed at controlling her disease including prednisone, methotrexate, hydroxyurea, photopheresis, interferon-α, PUVA, and cladarabine. After more than 10 years of receiving these standard therapies, the patient had persistent disease involving her skin, lymph nodes, peripheral blood, and bone marrow.

Bone marrow transplantation, once used only after failure of conventional types of therapy, is now the preferred therapy for many malignant hematologic disorders [19,20]. Allogeneic BMT has been most commonly used for the eradication of acute and chronic leukemia. For some hematologic diseases such as aplastic anemia, chronic myelogenous leukemia, myelodyplasia and acute leukemia in relapse, chronic lymphocytic leukemia, and leukemic phase of malignant lymphoma, allogeneic BMT from a matched sibling or unrelated donor is the only viable potentially curative therapeutic option.

Allogeneic BMT has been reserved primarily for patients with advanced or multiply recurrent "poor-risk" lymphoma who are not considered to be appropriate candidates for autologous transplantation [21–24]. Relapse rates after allogeneic BMT are substantially lower compared with autologus transplantation in patients with heavily pretreated low-grade lymphoma, most likely due to a graft-vs.-lymphoma (GVL) effect [22,25–27]. Patients receiving allogeneic BMT have a higher risk of treatment-related mortality, especially if an unrelated donor is used, but a lower risk of relapse and improved disease-free survival [27–29]. These studies suggest that allogeneic BMT may be potentially curative in selected patients with advanced lymphoma, particularly those with low-grade histologies [25–27,29]. Unlike results using autologous transplants, there are very few relapses after 3 years in this group of patients.

Kuzel *et al.* [30] have reported preliminary results of allogeneic BMT in two patients with tumor-stage MF who had failed a variety of topically directed and systemic therapies. Both patients were in remission at 200 and 101 days posttransplant at the time of this report. Further investigation on the use of allogeneic BMT in the treatment of patients with advanced MF/CTCL is warranted, inasmuch as a GVL effect is known to accompany GVHD in patients with lymphoma [22]. Because GVHD frequently occurs in the skin, the most common site of involvement of MF/CTCL, it is also intriguing to consider whether this phenomenon may contribute to the efficacy of BMT in the treatment of these disorders. In the patient presented in this report, there was no correlation between the distribution of cutaneous chronic GVHD and sites of previous lymphomatous skin involvement.

CSA, a potent inhibitor of T-cell activation and expansion, is commonly used for the prevention and treatment of GVHD after BMT. Cooper *et al.* [31] conducted a phase II study of CSA for the treatment of refractory T-cell lymphomas, including CTCL. The dose of CSA used in this study was higher (7.5 mg/kg twice a day) than the dose used in BMT, which is usually 5.0 mg/kg twice a day up to day 83, followed by tapering doses thereafter [11]. None of the patients with peripheral T-cell lymphoma responded, but two of 11 patients with CTCL achieved a temporary clinical complete response. In both patients, disease activity recurred within a week after withdrawal of therapy. These observations suggest that CSA exerted a cytostatic rather than a cytotoxic effect or, alternatively, that CSA treatment resulted in clinical improvement by decreasing an inflammatory response secondary to the CTCL cells. Although another response was seen after CSA was readministered at a lower dose, both patients eventually became resistant to CSA and had disease progression.

It is unlikely that the remission achieved by our patient is attributable to the use of CSA. The trial by Cooper *et al.* indicates that the response rate and duration of response to CSA are disappointingly low in CTCL. None of the patients in their study had evidence of Sezary syndrome. Moreover, the remission status in our patient has been documented by immunophenotypic and T-cell receptor gene rearrangement studies at 36 months post-BMT, and it is unlikely that this clinical and molecular remission could be achieved with an agent that is cytostatic rather than cytotoxic.

MF/CTCL generally has a long natural history and initially behaves as a low-grade neoplasm. Our patient had advanced disease for >10 years without ever having achieved a clinical remission before BMT despite multiple therapies. Based on this case and the experience of allogeneic BMT for other patients with low-grade lymphoma, we suggest that younger patients with advanced MF/CTCL and Sezary syndrome who fail standard therapies such as photopheresis or interferon-α should be considered for allogeneic BMT from a sibling or unrelated donor. Longer follow-up will be required to determine the durability of remission after BMT.

ACKNOWLEDGMENTS

The authors wish to thank Barbara Littrell, RN, nurse coordinator for the National Marrow Donor Program Office at the City of Hope National Medical Center, for assisting in the evaluation of this patient for unrelated donor transplantation. We also wish to thank Dr. Herschel Zackheim for providing clinical data and for helpful discussions regarding this patient.

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