E-CASE REPORT

Treatment of Sézary Syndrome With Alemtuzumab: A Series of 5 Cases and a Review of the Literature

E. del Alcázar-Viladomiu, A. Tuneu-Valls, A. López-Pestaña, M.J. Vidal-Manceño

Servicio de Dermatología, Hospital Universitario Donostia, San Sebastián, Spain
Servicio de Hematología, Hospital Universitario Donostia, San Sebastián, Spain

Received 24 September 2014; accepted 8 February 2015

KEYWORDS
Sézary syndrome; Alemtuzumab; Treatment

PALABRAS CLAVE
Síndrome de Sézary; Alemtuzumab; Tratamiento

Abstract
Alemtuzumab is a monoclonal antibody that has been used to treat refractory cases of Sézary syndrome (SS) and advanced mycosis fungoides.

We present 5 patients with SS who were treated with alemtuzumab between 2008 and 2012, with an overall response rate of 80% (40% partial response and 40% complete response). A regimen of 10 mg administered subcutaneously was well tolerated with acceptable toxicity. The median duration of response was 13 months. However, one patient remains in complete remission after 67 months, a remarkable outcome given the low survival rate associated with SS.

In conclusion, we believe that alemtuzumab may be useful in cases of SS refractory to other treatments. As there are no curative treatments for SS, alemtuzumab should be considered as a therapeutic option.

© 2014 Elsevier España, S.L.U. and AEDV. All rights reserved.

Tratamiento del síndrome de Sézary con alemtuzumab: serie de 5 casos y revisión de la literatura

Resumen
Alemtuzumab es un anticuerpo monoclonal que se ha utilizado como terapéutica en casos refractarios de síndrome de Sézary (SS) y micosis fungoide en estadio avanzado.

Presentamos 5 pacientes diagnosticados de SS tratados con alemtuzumab entre los años 2008 y 2012, con una tasa de respuesta global del 80% (40% respuestas parciales y 40% respuestas completas). La pauta de 10 mg vía subcutánea fue bien tolerada y con una toxicidad aceptable. En nuestra casuística la mediana de duración de la respuesta fue de 13 meses, sin embargo uno de los pacientes continúa en remisión completa tras 67 meses, hecho destacable dada la baja supervivencia del SS.

Please cite this article as: del Alcázar-Viladomiu E, Tuneu-Valls A, López-Pestaña A, Vidal-Manceño MJ. Tratamiento del síndrome de Sézary con alemtuzumab: serie de 5 casos y revisión de la literatura. Actas Dermosifiliogr. 2015;106:e33–e39.

* Corresponding author.
E-mail address: elenadelalcazarviladomiu@gmail.com (E. del Alcázar-Viladomiu).

1578-2190/© 2014 Elsevier España, S.L.U. and AEDV. All rights reserved.
Introduction

Sézary syndrome (SS) is a rare disorder that accounts for approximately 3% of all cutaneous T-cell lymphomas. It is considered to be an aggressive variant characterized by the presence of erythroderma and circulating atypical T cells (Sézary cells), with or without lymphadenopathy. 1 SS is classified as T4, N0-3, M0-1, B2 according to the International Society for Cutaneous Lymphomas (Table 1). 1-3 Pruritus is the main symptom and can have a marked impact on quality of life.

The prognosis is poor, with a 5-year survival of 24%; few lasting responses or remissions have been reported after treatment. 6

Systemic therapies are necessary for treatment, as those that exclusively target the skin are insufficient. The therapeutic choice is determined by the spread of the disease, the impact on quality of life, and the patient’s age and comorbid conditions. Although several treatments are available for SS (Table 2), 3 few efficacy studies have been published.

New therapies for SS, including biological therapies, have appeared in recent years. Alemtuzumab is a humanized monoclonal antibody that targets the CD52 glycoprotein expressed on the surface of T and B cells, natural killer cells, monocytes, and macrophages, leading to a depletion of those cells in the peripheral blood. 4,5 It is thought to act by direct cell lysis mediated by neutrophils, complement, and antibody-dependent cytotoxicity and apoptosis. 3-6 In 2001, alemtuzumab was approved for the treatment of B-cell chronic lymphocytic leukemia, although some authors have used it in cases of refractory SS and advanced mycosis fungoides (MF).

Case Descriptions

We present 5 patients (3 women and 2 men) diagnosed with stage IVA, SS (T4, N0, M0, B2) who were treated with alemtuzumab between 2008 and 2012. Four patients had undergone previous treatments (phototherapy, prednisone, methotrexate, gemcitabine, and bexarotene).

Data were gathered on the following variables to assess the treatment with alemtuzumab (Table 3): duration, type of response, time until onset of the response, duration of the response after the interruption of treatment, and peripheral blood Sézary cells. In addition, we analyzed the clinical course, follow-up period, and complications associated with alemtuzumab.

The type of response was classified as follows: complete response (CR), partial response (PR), or disease progression (DP), in accordance with the study by Olsen et al. 2 (Figure 1).

The regimen used was the subcutaneous administration of an initial dose of 3 mg, followed by doses of 10 mg 3 times per week.

The 5 patients received oral prophylaxis with sulfamethoxazole-trimethoprim, aciclovir, and fluconazole for Pneumocystis jiroveci, herpesvirus, and Candida. For cytomegalovirus (CMV) prophylaxis, we performed weekly measurement of the viral load and administered anticipatory treatment with oral valganciclovir in the event of reactivation.

A response was achieved in 4 of the 5 patients (overall response rate, 80%); there were 2 PR (40%) and 2 CR (40%). The median duration of treatment was 8 weeks (range, 4-13 weeks) and the median duration of the response after the interruption of treatment was 13 months (range, 5-66 months).

Complications included pneumococcal pneumonia in 1 patient, a cytokine release syndrome in 3, and subclinical CMV reactivation in 2. There were no hematological complications.

The median follow-up was 27 months (range, 23-67 months) after the initiation of treatment. One patient died at 24 months due to transformation to a high-grade non-Hodgkin lymphoma. At the time of writing, 3 patients are in progression and one continues in complete remission 67 months after completing the treatment.

Discussion

Alemtuzumab has been used in both refractory, advanced-stage MF and in SS. The first description is from 1998 in 8 patients with MF. 7 In 2003, the usefulness of alemtuzumab was demonstrated in a phase II study of 22 patients with MF or SS. 8 A number of small series and case reports have been published since that time. In the literature reviewed, we found a total of 13 series 4,6-17 (detailed in Table 4) and 6 case reports of MF and SS.

The overall responses to treatment in the 2 largest studies—of 22 and 39 patients—were 55% and 51%, respectively. 4,9 The median duration of the response was 12 months, although this varied between series. 4,6-17 In the series of 39 cases, lasting remissions (more than 2 years) were observed in 5 patients with SS but in only 1 patient with MF. 9

The treatment regimen has changed over the years. The drug was administered intravenously in the initial studies: sequential initial doses of 3 mg, 10 mg, and 30 mg were administered, followed by 30 mg 3 times a week. 7,8,10-13 It was subsequently observed that alemtuzumab could be equally effective administered subcutaneously at low doses. 6,14
Table 1  Classification of Mycosis Fungoides/Sézary Syndrome Proposed by the International Society for Cutaneous Lymphomas and the European Organisation for Research and Treatment of Cancer.

<table>
<thead>
<tr>
<th>Skin (T)</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1</td>
<td>Limited patches, papules, and/or plaques covering &lt;10% of the skin surface. T1a (patches only) and T1b (patches and plaques)</td>
</tr>
<tr>
<td>T2</td>
<td>Patches, papules, and/or plaques covering ≥10% of the skin surface. T2a (patches only) and T2b (patches and plaques)</td>
</tr>
<tr>
<td>T3</td>
<td>One or more tumors (≥1 cm in diameter)</td>
</tr>
<tr>
<td>T4</td>
<td>Confluent erythema covering ≥80% of the skin surface</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Nodes (N)</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>N0</td>
<td>No abnormal peripheral lymph nodes. Biopsy not required</td>
</tr>
<tr>
<td>N1</td>
<td>Clinically abnormal peripheral lymph nodes; histologically Dutch grade 1 or NCI LN1b-2. N1a, clone negative; N1b, clone positive</td>
</tr>
<tr>
<td>N2</td>
<td>Clinically abnormal peripheral lymph nodes; histologically Dutch grade 2 or NCI LN2a. N2a, clone negative; N2b, clone positive</td>
</tr>
<tr>
<td>N3</td>
<td>Clinically abnormal peripheral lymph nodes, histologically Dutch grade 3-4 or NCI LN4; clone positive or negative</td>
</tr>
<tr>
<td>Nx</td>
<td>Clinically abnormal lymph nodes without histological confirmation</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Visceral (M)</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>M0</td>
<td>No visceral involvement</td>
</tr>
<tr>
<td>M1</td>
<td>Visceral involvement (histological confirmation required and organ must be specified)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Blood (B)</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>B0</td>
<td>Absence of significant blood involvement (&lt;5% atypical lymphocytes [Sézary cells]). B0a, clone negative; B0b, clone positive</td>
</tr>
<tr>
<td>B1</td>
<td>Low blood tumor burden (≥5% atypical lymphocytes [Sézary cells]). B1a, clone negative; B1b, clone positive</td>
</tr>
<tr>
<td>B2</td>
<td>High blood tumor burden defined as one of the following: ≥1,000 Sézary cells/μL with positive TCR; CD4/CD8 &gt; 10 with positive TCR; or CD4+ CD7- cells ≥40% or CD4+ CD26- cells ≥30% with positive TCR</td>
</tr>
</tbody>
</table>

Abbreviations: LN, lymph node; NCI, National Cancer Institute; TCR, T-cell receptor. Source: Olsen et al.2

Table 2  Treatments Used in Sézary Syndrome.

<table>
<thead>
<tr>
<th>Type of therapy</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Immune modulators</strong></td>
<td>Interferon (IFN alfa and IFN gamma)</td>
</tr>
<tr>
<td></td>
<td>Retinoid (bexarotene)</td>
</tr>
<tr>
<td></td>
<td>Denileukin diftitox</td>
</tr>
<tr>
<td></td>
<td>Extracorporeal photopheresis</td>
</tr>
<tr>
<td><strong>Biologics</strong></td>
<td>Alectuzumab</td>
</tr>
<tr>
<td><strong>Radiotherapy</strong></td>
<td>Total body irradiation with electrons (electron beam)</td>
</tr>
<tr>
<td><strong>Combine treatments</strong></td>
<td>IFN alfa + phototherapy or retinoid</td>
</tr>
<tr>
<td></td>
<td>Retinoid + phototherapy</td>
</tr>
<tr>
<td></td>
<td>Extracorporeal photopheresis + IFN alfa or retinoid</td>
</tr>
<tr>
<td><strong>Systemic chemotherapy</strong></td>
<td>Methotrexate</td>
</tr>
<tr>
<td><strong>Monotherapy</strong></td>
<td>Pegylated doxorubicin</td>
</tr>
<tr>
<td><strong>Polychemotherapy</strong></td>
<td>Purine/pyrimidine analogues (fludarabine, 2-chlorodeoxyadenosine, deoxycoformycin, gemcitabine, forodesine)</td>
</tr>
<tr>
<td></td>
<td>Alkylating agents (chlorambucil, nitrogen mustard, cyclophosphamide, temozolomide)</td>
</tr>
<tr>
<td></td>
<td>Topoisomerase inhibitors (etoposide, pegylated doxorubicin)</td>
</tr>
<tr>
<td></td>
<td>Histone deacetylase inhibitors (vorinostat, romidepsin)</td>
</tr>
<tr>
<td></td>
<td>CHOP and CHOP-like</td>
</tr>
<tr>
<td><strong>HSCT</strong></td>
<td>Autologous</td>
</tr>
<tr>
<td></td>
<td>Allogeneic</td>
</tr>
<tr>
<td></td>
<td>Nonmyeloablative allogeneic</td>
</tr>
</tbody>
</table>

Abbreviations: IFN, interferon; HSCT, hematopoietic stem cell transplant. Source: modified from Jawed et al.3
A, Erythematous lesions on the abdomen of a patient with SS before starting treatment with alemtuzumab (case 3). B, In the same patient, resolution of the erythema 4 weeks after starting treatment with alemtuzumab.

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age</th>
<th>Treatment Duration</th>
<th>Time to Onset</th>
<th>Type of Response&quot;</th>
<th>Type of Cutaneous Response&quot;</th>
<th>Overall Response</th>
<th>Duration of the response</th>
<th>Complications</th>
<th>Clinical Course and Follow-up After Starting Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>F (1)</td>
<td>57</td>
<td>4 wk</td>
<td>4 wk</td>
<td>CR</td>
<td>CR</td>
<td>CR</td>
<td>66 mo</td>
<td>No</td>
<td>CR 67 mo</td>
</tr>
<tr>
<td>M (2)</td>
<td>65</td>
<td>8 wk</td>
<td>2 wk</td>
<td>CR</td>
<td>CR</td>
<td>CR</td>
<td>11 mo</td>
<td>Cytokine release syndrome</td>
<td>Death due to high-grade NHL 24 mo</td>
</tr>
<tr>
<td>F (3)</td>
<td>85</td>
<td>13 wk</td>
<td>4 wk</td>
<td>CR</td>
<td>PR</td>
<td>PR</td>
<td>5 mo</td>
<td>No</td>
<td>Retreatment at 4 wk with no improvement. Currently chlorambucil-prednisone 27 mo</td>
</tr>
<tr>
<td>M (4)</td>
<td>75</td>
<td>5 wk</td>
<td>1 wk</td>
<td>PR</td>
<td>CR</td>
<td>PR</td>
<td>15 mo</td>
<td>Cytokine release syndrome</td>
<td>Currently photophoresis 32 mo</td>
</tr>
<tr>
<td>F (5)</td>
<td>57</td>
<td>10 wk</td>
<td>No response</td>
<td>DP</td>
<td>CR</td>
<td>DP</td>
<td>Not recorded</td>
<td>Cytokine release syndrome</td>
<td>Allogeneic bone marrow transplant 20 mo</td>
</tr>
</tbody>
</table>

Abbreviations: CR, complete response; DP, disease progression; F, female; M, male; NHL, non-Hodgkin lymphoma; PR, partial response; X, median values.

CR: 100% lesion clearance; PR: 50%-99% clearance of initial lesions with no new tumors (T3) in patients with T1, T2, or T4; DP: ≥ 25% increase in skin disease or new tumors (T3) in patients with T1, T2, or T4, or loss of response in patients with complete response or partial response. F. CR: B0 and PR > 50% reduction in the initial blood tumor burden. B. Patient 4 presented < 1000 Sezáry cells but a CD4/CD8 ratio > 10 and was therefore considered to have stage IVA1 disease, as the others.
## Table 4  Published Series of Patients With SS/MF Treated With Alemtuzumab.

<table>
<thead>
<tr>
<th>Series</th>
<th>No. of Patients</th>
<th>Alemtuzumab Regimen</th>
<th>Treatment Duration</th>
<th>Clinical Response</th>
<th>Response Duration (Median)</th>
<th>Serious Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lundin J et al.(^7) (1998)</td>
<td>50 low grade NHL (8 MF)</td>
<td>30 mg IV on 3 d/wk</td>
<td>6-12 wk</td>
<td>OR 20% MF: CR 50%, 2 CR</td>
<td>10 mo</td>
<td>Opportunistic infections (7), sepsis (9), grade iv neutropenia (14)</td>
</tr>
<tr>
<td>Lundin J et al.(^8) (2003)</td>
<td>22 MF/SS</td>
<td>30 mg IV on 3 d/wk</td>
<td>10 wk</td>
<td>CR 55% 7 CR, 5 PR, 3 SD, 7 DP</td>
<td>12 mo</td>
<td>CMV reactivation (4), FUO (3), generalized HSV (1), pulmonary aspergillosis (1), Mycobacterium pneumonae (1), febrile neutropenia (1)</td>
</tr>
<tr>
<td>Kennedy GA et al.(^10) (2003)</td>
<td>5 MF/2 SS 1 MF transformed to large cell</td>
<td>30 mg IV on 3 d/wk</td>
<td>3-13 wk</td>
<td>OR 38% 3 PR, 2 SD, 3 DP</td>
<td>Less than 3 mo</td>
<td>Grade iv pancytopenia with sepsis (2), cutaneous MRSA and oral HSV (1), viral bronchiolitis (1), cutaneous VZV (1), CMV (1), Pseudomonas osteomyelitis and Parvovirus infection (1), sepsis due to Klebsiella (1)</td>
</tr>
<tr>
<td>Ferrajoli et al.(^11) (2003)</td>
<td>6 CTCL</td>
<td>30 mg IV on 3 d/wk</td>
<td>4-12 wk</td>
<td>2 PR</td>
<td>NS</td>
<td>No</td>
</tr>
<tr>
<td>Capalbo S et al.(^12) (2003)</td>
<td>1 MF/2 SS</td>
<td>30 mg IV on 3 d/wk</td>
<td>3, 6, and 12 wk</td>
<td>1 CR, 1 PR, 1 death (AMI)</td>
<td>11 mo</td>
<td>Cardiovascular events (5): CHF/arrhythmia Two deaths from unspecified infections</td>
</tr>
<tr>
<td>Lenihan DJ et al.(^13) (2004)</td>
<td>5 SS and 3 MF</td>
<td>30 mg IV on 3 d/wk</td>
<td>8.5 wk</td>
<td>3 PR, 2 SD, 3 DP</td>
<td>NS</td>
<td>One Legionella pneumonae pneumonia CMV reactivation (1)</td>
</tr>
<tr>
<td>Zinzani et al.(^14) (2005)</td>
<td>4 MF 6 peripheral TCL 14 SS</td>
<td>10 mg SC 3 d/wk</td>
<td>4 wk</td>
<td>3 PR (MF)</td>
<td>NS</td>
<td>Infections in patients treated with 15 mg: Staphylococcal sepsis (1)</td>
</tr>
<tr>
<td>Bernengo et al.(^4) (2007)</td>
<td>5 SS</td>
<td>10-15 mg SC 3 d/wk</td>
<td>NS</td>
<td>1 CR, 11 PR, 2 SD</td>
<td>12 mo</td>
<td>Subclinical CMV reactivation (3) Asymptomatic CMV reactivation (2), EBV reactivation (1)</td>
</tr>
<tr>
<td>Alinari et al.(^15) (2007)</td>
<td>30 mg SC 3 d/wk</td>
<td>5-9 wk</td>
<td>OR 100%</td>
<td>5 CR</td>
<td>8 wk</td>
<td>Asymptomatic CMV reactivation (2), EBV reactivation (1)</td>
</tr>
</tbody>
</table>
Table 4  (Continued)

<table>
<thead>
<tr>
<th>Series</th>
<th>No. of Patients</th>
<th>Alemtuzumab Regimen</th>
<th>Treatment Duration</th>
<th>Clinical Response</th>
<th>Response Duration (Median)</th>
<th>Serious Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Querfeld et al., 16 (2009)</td>
<td>17 SS</td>
<td>2 erythrodermic MF</td>
<td>30 mg IV and SC, 3 d/wk</td>
<td>12 wk</td>
<td>OR 84% 9 CR, 7 PR, 3 DP</td>
<td>6 mo</td>
</tr>
<tr>
<td>Clark level et al, 17 (2012)</td>
<td>18 CTCL-L</td>
<td>10 mg SC</td>
<td>30 mg IV and SC, 3 d/wk</td>
<td>6 wk</td>
<td>OR 89% 9 CR, 7 PR, 2 DP</td>
<td>NS</td>
</tr>
<tr>
<td>De Masson et al. 17 (2014)</td>
<td>23 SS/16 MF</td>
<td>10 mg SC</td>
<td>30 mg IV and SC, 3 d/wk</td>
<td>12 wk</td>
<td>NR 51% (70% SS, 25% MF)</td>
<td>NS</td>
</tr>
<tr>
<td>Watanabe et al. 17 (2014)</td>
<td>17 SS/6 MF</td>
<td>10 mg SC</td>
<td>NS</td>
<td>10 mg SC</td>
<td>SS: 13 CR, 4 PR MF: 1 CR after electron beam, 5DP</td>
<td>NS</td>
</tr>
<tr>
<td>Our series</td>
<td>5 SS</td>
<td>10 mg SC</td>
<td>8 wk</td>
<td>12 wk</td>
<td>2 CR, 2 PR, 1 DP</td>
<td>13 mo</td>
</tr>
</tbody>
</table>

Abbreviations: AMI, acute myocardial infarction; CBCL, cutaneous B-cell lymphoma; CHF, congestive heart failure; CMV, cytomegalovirus; CR, complete response; CTCL, cutaneous T-cell lymphoma; CTCL-L, leukemic cutaneous T-cell lymphoma; DP, disease progression; EBV, Epstein Barr virus; FUO, fever of unknown origin; HSV, herpes simplex virus; MRSA, methicillin-resistant *Staphylococcus aureus*; NHL, non-Hodgkin lymphoma; OR, overall response; PR, partial response; SC, subcutaneous; SD, stable disease; TB, tuberculosis; TCL, T-cell lymphoma; VZV, varicella-zoster virus.

Treatment-related complications include infection and hematologic toxicity. The regimen of 10 mg subcutaneously has been shown to be associated with a lower incidence of these side effects. 4,14,15 The most common opportunistic infection is CMV reactivation, although other types of infection have also been reported. 4 Myelosuppression is the most common hematologic toxicity. 4

Cytokine release syndrome is the most common complication during treatment. 7,8,10,11 Its frequency can be reduced by progressive dose escalation. Lenihan et al. 11 published 4 cases of cardiac toxicity during the treatment of previously healthy patients. As this has not been observed in any other study, the possibility of a causal relationship remains under debate. A case of cutaneous hemophagocytosis at the site of injection of alemtuzumab was recently reported in a patient without Epstein-Barr virus reactivation. 18

In the literature reviewed, alemtuzumab has been used indistinctly for MF and for SS, and advanced-stage SS and MF are grouped together in most studies. Erythrodermic MF is now considered to be progression of MF with absent or minimal blood involvement, in contrast to the situation with SS, which arises de novo and shows significant blood involvement. 1 In 2012, Clark et al. 3 reported that alemtuzumab may only be effective when blood involvement is present (in SS and in cases of erythrodermic MF with blood involvement). Those authors suggested that the 2 diseases
arose from distinct types of memory T cells: the malignant lymphocytes in patients with SS have a CCR7+/L-selectin−
central memory cell phenotype (migratory cells that are
found in peripheral blood, in the skin, and in lymph nodes),
whereas the malignant lymphocytes of MF arise from non-
migratory cells resident in the skin and that are not found in
the peripheral blood. Alemtuzumab depletes all T lympho-
cytes in the blood, but the population of cells resident
in the skin will escape from the effect of the antibody and
persist after treatment. In addition, alemtuzumab requires
the presence of neutrophils and natural killer cells (both of
which are present in blood but not in skin) to achieve lym-
phocyte depletion, and hence it only eliminates cells in the
peripheral blood. The main candidates for this treatment
are therefore patients with SS.

Unfortunately, authorization for the indication of this
drug in B-cell chronic lymphocytic leukemia and other hema-
tological diseases was withdrawn in August 2012, and it is
currently only available for multiple sclerosis. For the treat-
ment of patients with SS, alemtuzumab can only be obtained
by individualized access via protocol.

Conclusions

We have presented 5 cases of SS treated with alemtuzumab,
achieving an 80% response rate. The regimen of 10 mg
administered subcutaneously was well tolerated and the
median duration of the response was 13 months. Alem-
tuzumab can be a useful drug in cases of SS refractory to
other treatments, achieving a rapid clinical response and
improvement in quality of life, with a reduction or remission
of the pruritus.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Ethical Disclosures

Protection of human and animal subjects. The authors
declare that no experiments were performed on humans or
animals for this research.

Confidentiality of data. The authors declare that no private
patient data are disclosed in this article.

Right to privacy and informed consent. The authors
declare that no private patient data are disclosed in this
article.

References

1. Jawed SI, Myskowski PL, Horwitz S, Moskowitz A, Querfeld
   C. Primary cutaneous T-cell lymphoma (mycosis fungoides and
   Sézary syndrome): Part I diagnosis: Clinical and histopathologic
   features and new molecular and biologic markers. J Am Acad
   SR, et al. Clinical end points and response criteria in mycosis
   fungoides and Sézary syndrome: A consensus statement of the
   International Society for Cutaneous Lymphomas, the United
   States Cutaneous Lymphoma Consortium, and the Cutaneous
   Lymphoma Task Force of the European Organisation for
3. Jawed SI, Myskowski PL, Horwitz S, Moskowitz A, Querfeld
   C. Primary cutaneous T-cell lymphoma (mycosis fungoides and
   Sézary syndrome): Part II. prognosis, management, and future
4. Bernengo MG, Quaglino P, Comessatti A, Ortonelli M, Novelli M,
   Lisa F, et al. Low-dose intermittent alemtuzumab in the treat-
   ment of Sézary syndrome: Clinical and immunologic findings in
5. Izu-Belloso RM, Garcia-Ruiz JC. Actualización terapéutica en
6. Clark RA, Watanabe R, Teague JE, Schlapbach C, Tawa MC,
   Adams N, et al. Skin effector memory T cells do not recircu-
   late and provide immune protection in alemtuzumab-treated
7. Lundin J, Osterborg A,Brittiger G, Crowther D, Dombret H,
   Engert A, et al. CAMPATH-1H monoclonal antibody in therapy
   for previously treated low-grade non-Hodgkin’s lymphomas: A
   phase II multicenter study European Group of CAMPATH-
8. Lundin J, Hagberg H, Repp R, Cavallin-Stahl E, Fredén S,
   Juliusson G, et al. Phase 2 study of alemtuzumab (anti-CD52
   monoclonal antibody) in patients with advanced mycosis fun-
   Bouaziz JD, et al. Long-term efficacy and safety of alemtuzumab
10. Kennedy GA, Seymour JF, Wolf M, Januszewicz H, Davison J,
    McCormack C, et al. Treatment of patients with advanced myco-
    sis fungoides and Sézary syndrome with alemtuzumab. Eur J
    S, et al. Phase II study of alemtuzumab in chronic lympho-
12. Capalbo S, Delia M, Dargenio M, Liso A, Diomede D, Garofalo
    cases treated with Campath-1H as salvage treatment. Med
    M. Cardiac toxicity of alemtuzumab in patients with mycosis
    Preliminary observations of a phase II study of reduced-dose
    alemtuzumab treatment in patients with pretreated T-cell
15. Alinari L, Gelskin L, Grady T, Baiocchi RA, Bechtel MA, Porcu
    P. Subcutaneous alemtuzumab for Sézary syndrome in the very
    P, et al. Alemtuzumab for relapsed and refractory erythroder-
    mic cutaneous T-cell lymphoma: A single institution experience
    from the Robert H Lurie Comprehensive Cancer Center. Leuk
17. Watanabe R, Teague JE, Fisher DC, Kupper TS, Clark RA. Alem-
    tuzumab therapy for leukemic cutaneous T-cell lymphoma:
    Diffuse erythema as a positive predictor of complete remission.
    hemophagocytosis after alemtuzumab injection in a patient with