Correspondence to: Choon Chiat Oh, MD, Singapore General Hospital, Department of Dermatology, Academia Level 4, 20 College Rd, Singapore 169856

E-mail: oh.choon.chiat@singhealth.com.sg

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$CD30^+$ leukemic cutaneous T-cell lymphoma

To the Editor: CD30 is a tumor necrosis factor receptor involved in regulating apoptosis and proliferation of $CD30^+$ tumor cells.[1] $CD30^+$ T cells are primarily expressed in conditions such as primary cutaneous $CD30^+$ T-cell lymphoproliferative disorders and some peripheral T-cell lymphomas, and they can potentially be expressed in any type of cutaneous T-cell lymphoma (CTCL).[2-4] However, CD30 expansion on the leukemic cells of patients with CTCL or Sézary syndrome is rare, and little is known regarding the natural history and prognosis of patients with leukemic CTCL expressing this cell type.[2,3,5]

After approval by an institutional review board, we identified all patients with a clinical diagnosis of leukemic CTCL or Sézary syndrome and reviewed the medical records of those exhibiting CD30 expression on the skin, in lymph nodes, or in blood from January 1, 1994, to August 31, 2017. Among 44 patients diagnosed with leukemic CTCL, 6 (13.6%) were found to have some degree of CD30 expression on the leukemic cells. Only 3 of the 6 patients had complete records available for review.

Patient 1, a 57-year-old white man, presented with a 1-year history of a generalized folliculocentric eruption unresponsive to topical steroids. Blood flow cytometry (FC) showed an abnormal population of T cells (10% of all events), phenotypically $CD2^+$, $CD3^+$, $CD4^+$, $CD8^-$, $CD5^+$, CD7 partial, CD25 dim/negative, $CD26^-$, CD30 partial (Fig 1, *A-D*). The $CD4^{hi}$ (nonmalignant, red) cells, which are $CD7^+$ (Fig 1, *B*) and $CD26^+$ (Fig 1, *C*), are $CD30^-$ or very dim (Fig 1, *D*) relative to the CD4 dim (blue) malignant population. The patient was diagnosed with mycosis fungoides with B1 involvement and treated with extracorporeal photopheresis (ECP) and narrowband ultraviolet B, with significant improvement. His disease has continued to remain well controlled 5 years after the initial diagnosis.

Patient 2, a 55-year-old man, had Sézary cells on peripheral smear and blood FC showing an abnormal population of T cells (83% of all events), phenotypically $CD2^+$, $CD3^+$, $CD4^-$, $CD8^-$, $CD5^+$, CD7 partial, $CD25^-$, $CD26^-$, TCR$\alpha\beta^+$, and a small portion of $CD30^+$ cells (Fig 1, *E*). He was diagnosed with stage IV Sézary syndrome and despite treatment with vorinostat and ECP, he died 6 years after presentation.

Patient 3, a 77-year-old white man with blood FC showing an abnormal population of T cells (approximately 14% of all events), phenotypically CD2 dim, $CD3^+$, $CD4^+$, $CD8^-$, $CD5^+$, $CD7^+$, CD25 dim, $CD26^-$ (16%), and a partial portion of $CD30^+$ cells (Fig 1, *F*). He was diagnosed with mycosis fungoides and received several treatments including ECP, bexarotene, and vorinostat. After several years of treatment, the patient self-discontinued therapy and died 7 years after presentation.

Because of challenges in the treatment of leukemic CTCL, it is important to understand its phenotypic variants and their implications for prognosis and treatment response. There has been little to no information on the expression of $CD30^+$ T cells in the blood of patients with leukemic CTCL. To our knowledge, CD30 is not routinely assessed on blood FC. Recent developments in the treatment of CTCL include antibody-based immunotherapies, including anti-CD30 brentuximab vedotin, currently approved for treatment of $CD30^+$ Hodgkin lymphoma, $CD30^+$ lymphoproliferative disorders, and CD30-expressing mycosis fungoides.[5,6] However, the nature of patients treated with brentuximab vedotin who have circulating $CD30^+$ cells is not yet clear.[5] Therefore, a better understanding of the role of leukemic CD30 positivity in the history, prognosis, and association with other prognostic factors and its significance in the development of more specific antineoplastic therapies may be beneficial to the treatment of patients with leukemic CTCL.

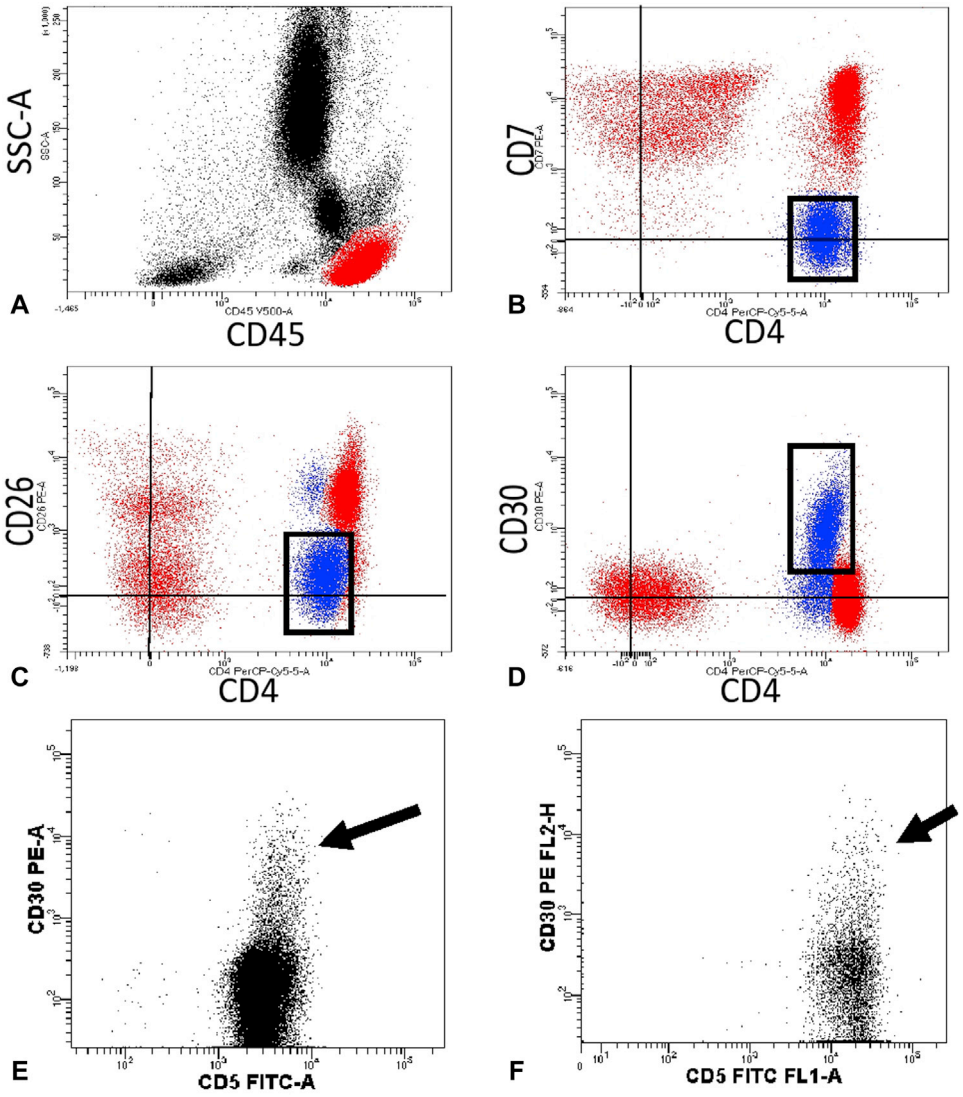

Fig 1. A-D, Patient 1. Red represents normal lymphocytes, and blue indicates leukemic (Sézary) cells. Cells are $CD7^{-}$ and $CD26^{-}$ (small subpopulation positive) with slightly decreased expression of CD4. CD30 expression is shown in the lower right dot plot. A total of 100,000 events were acquired on a FACSCanto flow cytometer (BD Biosciences, San Jose, CA) analyzed with DIVA software (BD Biosciences, San Jose, CA). **E**, Patient 2 and (**F**) patient 3, each exhibiting partial expression of CD30 on the $CD5^{+}$ gated leukemic (Sézary) population.

Ariana Ellis, BS,[a,b] Luisa F. Christensen, MD,[b] Timmie Sharma, MD,[b] Howard Meyerson, MD,[c] Honda Kord, MD,[b] and Kevin D. Cooper, MD[b]

From Northeast Ohio Medical University, Rootstown, Ohio[a]; Department of Dermatology, University Hospitals Cleveland Medical Center/Case Western Reserve University School of Medicine, Cleveland, Ohio[b]; Department of Pathology, University Hospitals Case Medical Center and Seidman Cancer Center Case Western Reserve University, Cleveland, Ohio.[c]

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Correspondence to: Ariana Ellis, BS, Northeast Ohio Medical University, 5901 Sharon Dr, Boardman, OH 44512

E-mail: aellis1@neomed.edu

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The association of frontal fibrosing alopecia with skin and hair care products: A survey-based case series of 56 patients seen at the Mayo Clinic

To the Editor: Frontal fibrosing alopecia (FFA) has become increasingly reported in the literature.[1-4] Studies have suggested an association between FFA and the use of facial skincare products and sunscreens.[1,2]

We identified 148 female and 7 male patients with FFA who were evaluated at the Mayo Clinic between 1992 and 2016.[4] Of the 155 patients who were invited, 56 (36.1%) completed the survey.

The survey design was based on previous FFA studies, particularly those suggesting environmental/sunscreen etiologies, as well as expert opinion (RT, ST). A retrospective chart review was performed. Statistical analysis was performed using the JMP Pro statistical software package, version 13.0 (SAS Institute Inc, Cary, NC). Descriptive statistics are reported as a number and percentage for discrete variables. Comparisons were evaluated using Fisher's exact test. All *P* values less than .05 were considered statistically significant.

Table I. Patient characteristics at presentation

Patient characteristics	Results
Age at diagnosis, y	
Mean	62.9
Median	64
Range	35-84
Age at onset of symptoms, y	
Mean	60.4
Median	63
Range	34-81
Sex, n (%)	
Female	56 (100)
Male	0 (0)
Race, n (%)	
White	55 (98.2)
Black	1 (1.8)
Geographic distribution, n (%)	
Midwest	47 (83.9)
South	4 (7.1)
Southwest	2 (3.6)
East Coast	2 (3.6)
Puerto Rico	1 (1.8)
Natural hair color, n (%)	
Brown	41 (73.2)
Blonde	10 (17.9)
Black	4 (7.1)
Red	1 (1.8)
Hair texture before hair loss, n (%)	
Fine or thin	19 (33.9)
Medium (neither thin nor thick)	19 (33.9)
Thick or coarse	18 (32.1)
Clinical examination findings, n (%)	
Eyebrow loss	45 (80.4)

Patient demographics are described in Table I. Most patients (89.3%) received a biopsy, with 100% showing histopathologic features consistent with FFA. Eyebrow loss was noted in 45 (80.4%) patients. Forty-four (78.6%) patients received continuous follow-up care at the Mayo Clinic. Response to treatment included unaltered disease progression in 20 (45.5%), slowing of disease progression in 17 (38.6%), and disease stabilization in 7 (15.9%).

Patients were asked to respond based on their behavior in the 5 to 10 years before the onset of hair loss. The results are summarized in Table II. The overall results indicate that 62.5% (n = 35) of patients with FFA used some facial sunscreen product daily. This rate is higher than that reported for regular sunscreen use across the United States (42.6% of women reported regularly using sunscreen on the face when outside on a warm sunny day for longer than 1 hour).[5] Additionally, 57.1% (n = 32) of patients with FFA reported regular (at least weekly)