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Case Report

Sézary Syndrome Presenting with Erythroderma and Circulating Cerebriform Lymphocytes: Diagnostic Contribution of Blood Cytology and Flow Cytometry – A Case Report and Literature Update

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Abstract: ***Background*:** Sézary syndrome (SS) is a rare, aggressive leukemic variant of cutaneous T-cell lymphoma (CTCL), defined by erythroderma, lymphadenopathy and circulating malignant T lymphocytes with cerebriform nuclei (Sézary cells). Its non-specific skin presentation often delays diagnosis. ***Case Presentation*:** A 55-year-old man with no relevant medical history presented with a generalized pruritic erythroderma evolving over several weeks, with asthenia and intermittent fever. Examination revealed cervical and axillary lymphadenopathy and mild splenomegaly (2 cm below the costal margin). ***Diagnostic Findings*:** The blood count showed moderate leukocytosis (12.77 G/L) without anemia (hemoglobin 12.8 g/dL) or thrombocytopenia (platelets 290 G/L). The May-Grünwald-Giemsa-stained blood smear showed 53% small-to-medium lymphoid cells with grooved, cerebriform nuclei. Whole-blood flow cytometry identified an abnormal T-cell population representing 84% of lymphocytes, with strong CD45, a CD3+/CD4+/CD7−/CD56− phenotype and a CD4/CD8 ratio >10. ***Conclusions*:** The association of erythroderma, characteristic Sézary cell morphology and an aberrant CD4+/CD7− phenotype allowed rapid diagnosis of SS. Combined blood smear review and flow cytometry are rapid, minimally invasive and decisive tools in the work-up of unexplained erythroderma.

Keywords: Sézary syndrome; cutaneous T-cell lymphoma; erythroderma; Sézary cells; flow cytometry.

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1. INTRODUCTION

Primary cutaneous T-cell lymphomas (CTCL) are a heterogeneous group of non-Hodgkin lymphomas that arise in the skin. Sézary syndrome (SS) is their most aggressive leukemic form and accounts for approximately 3–5% of all CTCL [1]. It is classically defined by generalized erythroderma involving more than 80% of the body surface area, superficial lymphadenopathy and more than 1 G/L circulating clonal T lymphocytes with cerebriform nuclei (Sézary cells).

Because erythroderma has many benign causes (generalized eczema, erythrodermic psoriasis, drug reactions), diagnosis depends on integrating clinical, cytomorphological and immunophenotypic data. We report a newly diagnosed case of SS in which blood smear examination and flow cytometry established the diagnosis, and we briefly review current diagnostic criteria and therapeutic options.

2. CASE PRESENTATION

2.1 Patient Information and Clinical Findings

A 55-year-old man with no notable medical history consulted for a generalized pruritic erythroderma that had progressed over several weeks, accompanied by deterioration of general condition, profound asthenia and intermittent fever.

Examination showed universal erythroderma with diffuse skin infiltration, bilateral, mobile, painless cervical and axillary lymphadenopathy, and mild splenomegaly palpable 2 cm below the costal margin. There was no hepatomegaly and no sign of major cutaneous superinfection.

2.2 Laboratory Investigations

The complete blood count showed leukocytes 12.77 G/L, hemoglobin 12.8 g/dL and platelets 290 G/L.

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Blood Smear:

Review of the May-Grünwald-Giemsa-stained smear showed 53% atypical lymphoid cells of small to medium size with moderately abundant basophilic cytoplasm lacking azurophilic granules. Nuclei had very irregular contours with deep grooves and notches, giving the cerebriform appearance typical of Sézary cells (Figures 1 and 2). This corresponds to an absolute count of approximately 6.8 G/L (53% × 12.77 G/L).

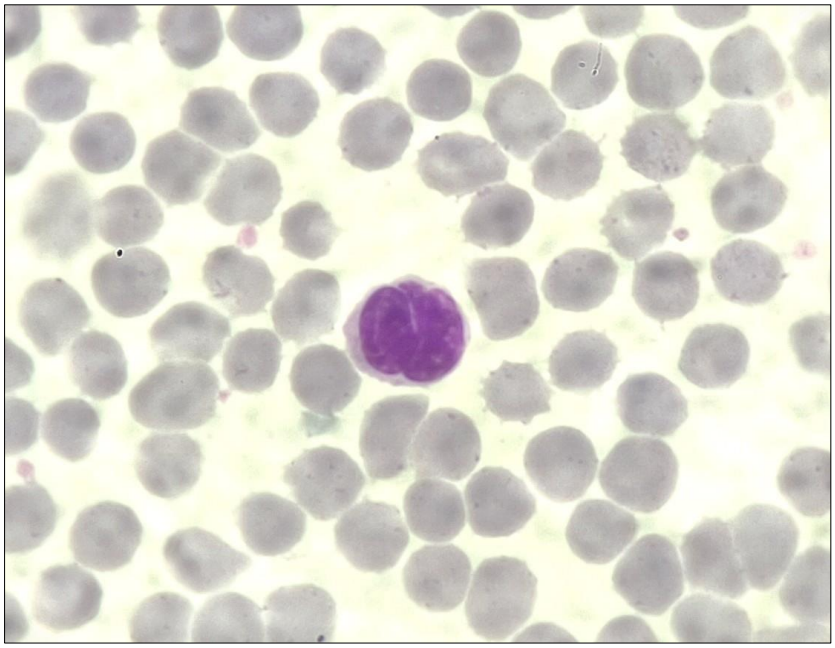

Figure 1: May-Grünwald-Giemsa-stained peripheral blood smear (×1000): Sézary cell.

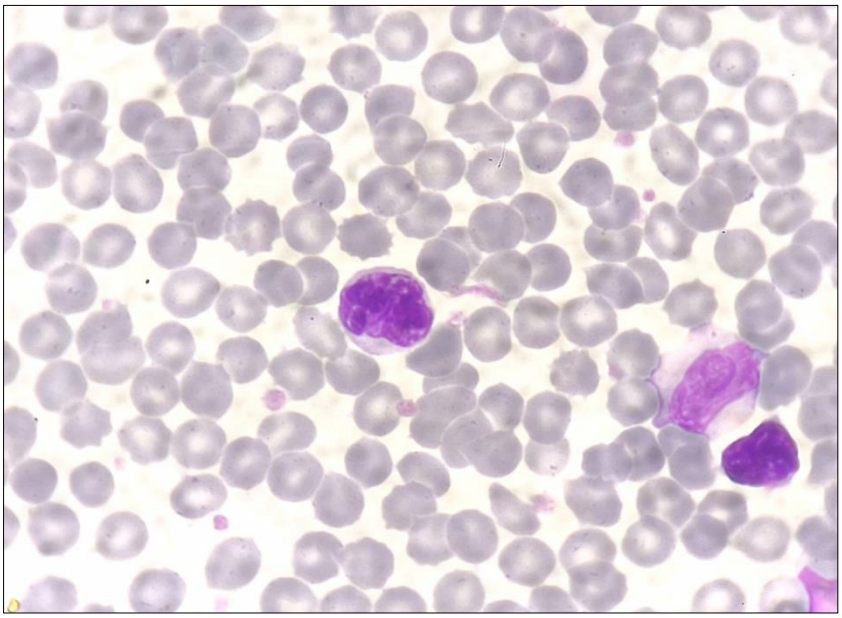

Figure 2: May-Grünwald-Giemsa-stained peripheral blood smear (×1000): Sézary cell.

Flow Cytometry:

Multiparameter flow cytometry on whole blood showed an abnormal T-cell population representing 84% of lymphocytes (Figures 3–5), with strong CD45, CD3+, CD4+, CD7− and CD56− expression and a CD4/CD8 ratio >10. Absence of CD56 argued against an NK-cell proliferation or T/NK lymphoma.

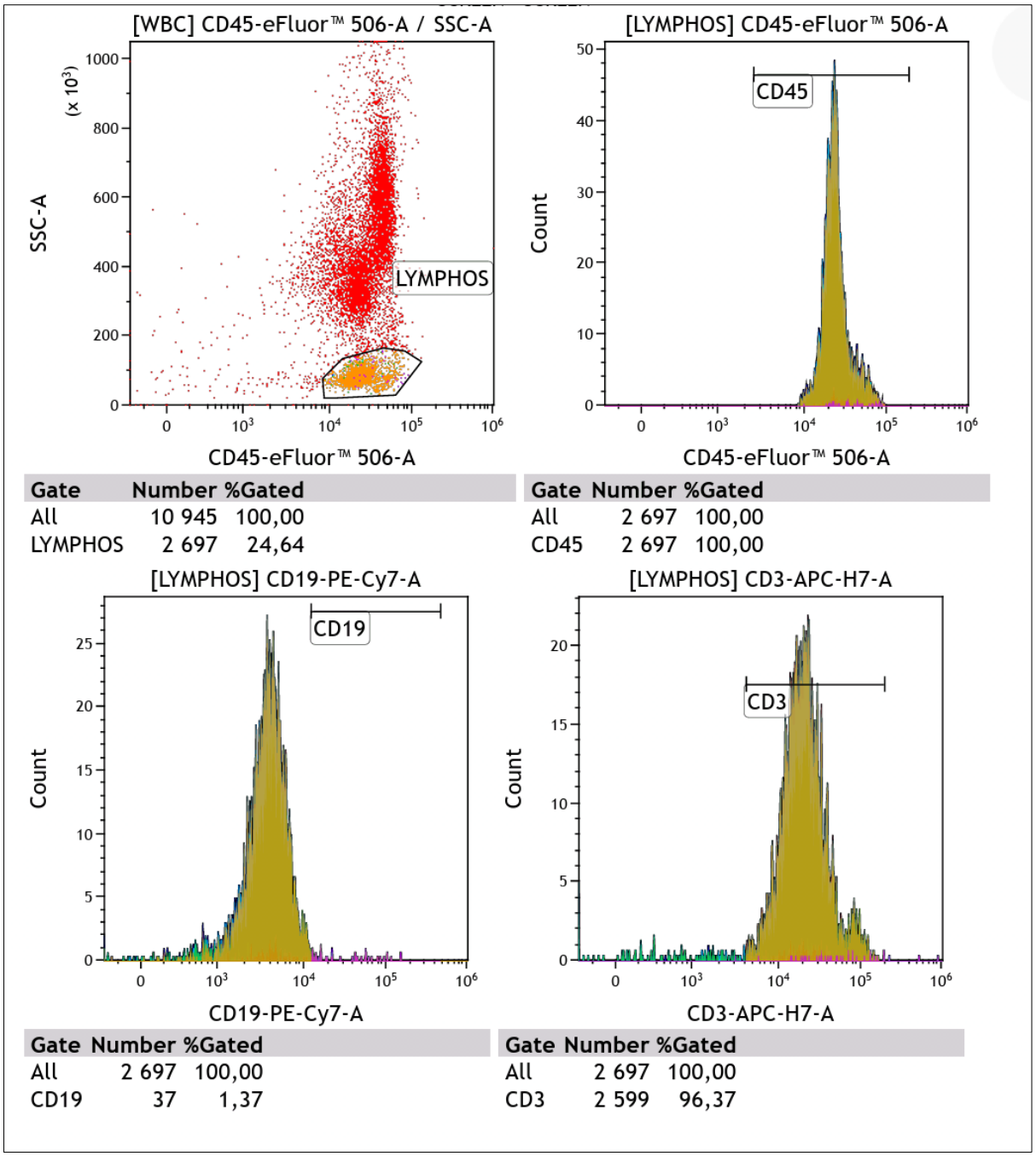

Figure 3: Lymphocyte gating showing predominance of an abnormal T-cell population.

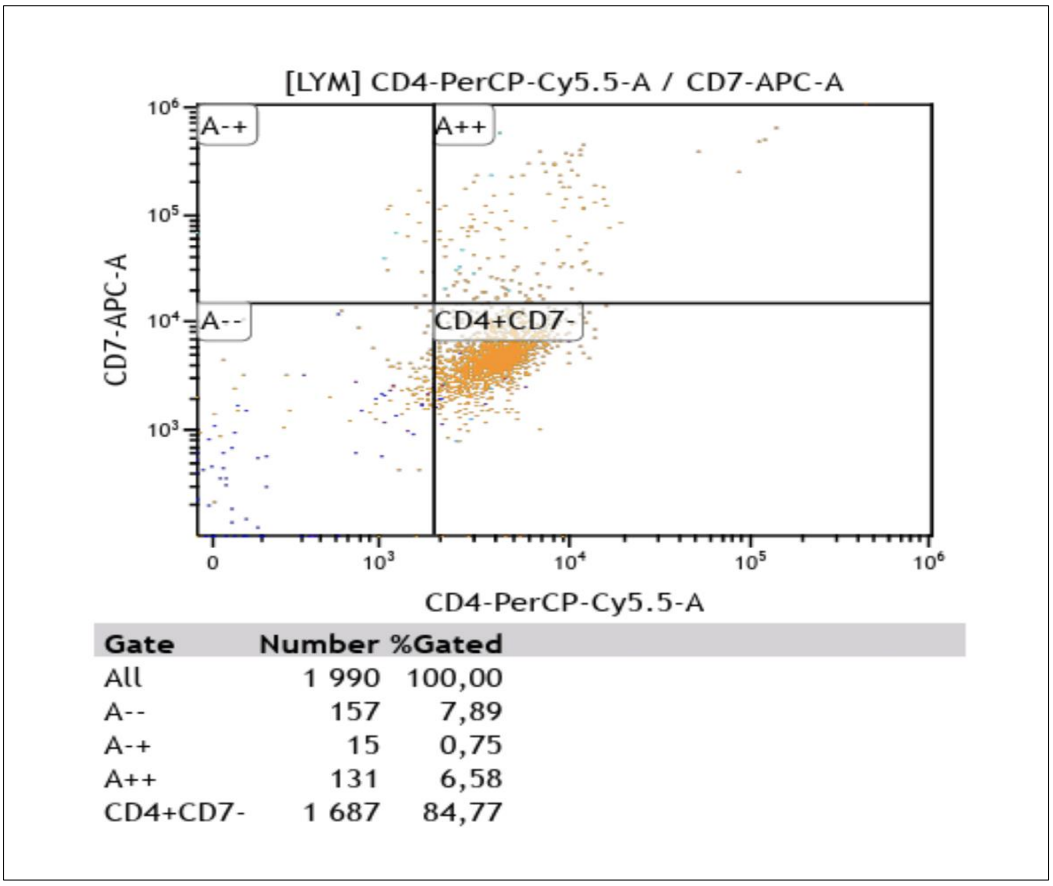

Figure 4: Abnormal population with CD3+/CD4+/CD7− phenotype.

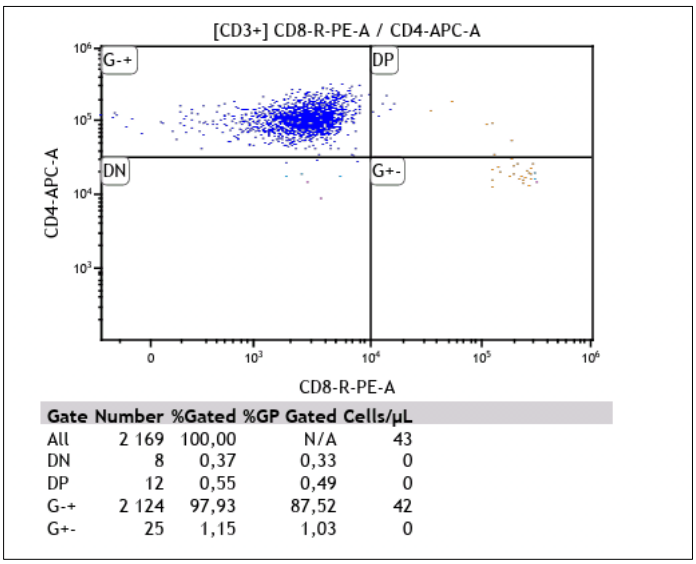

Figure 5: Predominance of CD4 population and CD4/CD8 ratio >10.

2.3 Diagnosis and Staging

The combination of erythroderma, a high proportion of circulating cerebriform cells and a CD3+/CD4+/CD7− phenotype fulfilled the ISCL/EORTC criteria for SS (Table 1).

Table 1: ISCL/EORTC diagnostic criteria applied to the present case

Criterion	ISCL/EORTC requirement	Present case
Skin involvement	Erythroderma	Universal pruritic erythroderma
Circulating Sézary cells	Absolute count >1 G/L	53% of leukocytes (≈6.8 G/L, calculated from 12.77 G/L)
CD4/CD8 ratio	>10	>10 (Figure 5)

Criterion	ISCL/EORTC requirement	Present case
Loss of pan-T antigens	CD7 and/or CD26	CD3+/CD4+/CD7− (84% of lymphocytes)
Clonal TCR rearrangement	Demonstrated clone	Not done

3. DISCUSSION

3.1 Principal Findings

This patient presented with the classic triad of SS: erythroderma, lymphadenopathy and a high burden of circulating Sézary cells. The diagnosis rested on rapidly available laboratory tests, namely blood smear morphology and whole-blood flow cytometry.

3.2 Diagnostic Criteria

According to ISCL/EORTC criteria, SS is defined by erythroderma together with at least one of the following: an absolute Sézary cell count above 1 G/L; an aberrant immunophenotype with a CD4/CD8 ratio above 10 or loss of T-cell antigens, notably CD7 or CD26; or a clonal T-cell receptor (TCR) gene rearrangement [2, 3]. Our patient met the first two criteria (Table 1).

Loss of surface pan-T antigens, particularly CD7 and CD26, is the most constant and clinically useful phenotypic abnormality. CD4 positivity with CD7 loss increases diagnostic confidence when facing benign inflammatory erythroderma such as generalized eczema or erythrodermic psoriasis, and absence of CD56 helps exclude rarer entities such as large granular lymphocyte leukemia or blastic T/NK lymphoma [3, 4].

3.3 Pathophysiology

Sézary cells are malignant helper memory T lymphocytes (CCR4+, CLA+) with marked skin tropism. Profound impairment of cellular immunity predisposes to cutaneous and systemic infections, which may account for the intermittent fever in our patient, although this was not formally investigated. At the molecular level, SS shows complex genomic instability, alterations of tumor suppressor genes (such as *TP53* and *PTEN*) and aberrant activation of JAK/STAT signaling [4].

3.4 Management

Because of early hematological dissemination, management of SS is systemic and multidisciplinary, involving hematologists and dermatologists [5]. Extracorporeal photopheresis is often used first-line or in combination, particularly in purely erythrodermic forms. Mogamulizumab, an anti-CCR4 antibody, targets malignant skin and circulating T cells and improved progression-free survival over vorinostat in the MAVORIC trial [5]. Other options include retinoids (bexarotene), interferons and histone deacetylase inhibitors (vorinostat). Allogeneic hematopoietic stem cell transplantation is the only potentially curative option and is reserved for eligible patients with refractory or advanced disease.

4. CONCLUSION

This case illustrates the typical presentation of Sézary syndrome in a 55-year-old man. Identification of cerebriform lymphocytes on the blood smear, combined with a CD4+/CD7− phenotype on flow cytometry, enables early diagnosis and timely initiation of appropriate treatment. Laboratory physicians should consider SS in any unexplained erythroderma with lymphadenopathy.

Key Learning Points

- Unexplained erythroderma with lymphadenopathy warrants a blood smear review for cerebriform lymphocytes.
- Loss of CD7 (and/or CD26) on CD4+ T cells and a CD4/CD8 ratio >10 by flow cytometry support the diagnosis and help distinguish SS from benign inflammatory erythroderma.
- Early recognition allows prompt referral for systemic, multidisciplinary management.

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