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Systemic Lupus Erythematosus and Acute Myeloid Leukemia: Coexistence, Diagnosis and Therapeutic Challenges

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ABSTRACT

Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease affecting multiple organ systems and characterized by systemic inflammation. Acute myeloid leukemia (AML) is an aggressive hematological malignancy marked by the clonal proliferation of immature myeloid cells in the bone marrow.

Case presentation

We report the case of a 35-year-old male with a known diagnosis of SLE who presented with general deterioration including fatigue, anorexia, a 15 kg weight loss over two months and fever (38.5°C), along with a perianal abscess. Laboratory investigations revealed pancytopenia with a hemoglobin level of 5.4 g/dL, WBC count of 2,600/mm³ and platelets at 16,000/mm³. Peripheral blood smear showed 55% blast cells. Bone marrow aspiration confirmed AML with Auer rods and translocation t(8;21). Management included empirical antibiotic therapy, antifungal prophylaxis, corticosteroids for SLE flare, followed by referral to hematology for chemotherapy. The patient responded well clinically and biologically.

Conclusion

The coexistence of SLE and AML is rare and poses complex diagnostic and therapeutic challenges. This case emphasizes the importance of a multidisciplinary approach and heightened clinical vigilance.

Keywords: Systemic lupus erythematosus; Acute myeloid leukemia; Diagnosis; Prognosis

INTRODUCTION

Systemic lupus erythematosus (SLE) is a multifaceted autoimmune disorder involving chronic systemic inflammation and multi-organ involvement. In contrast, acute myeloid leukemia (AML) is an aggressive hematologic malignancy resulting from the proliferation of undifferentiated myeloid precursors. Though pathogenetically distinct, their coexistence in a single patient presents diagnostic ambiguity and therapeutic complexity.

CASE PRESENTATION

A 35-year-old male with a history of SLE presented with systemic symptoms including marked fatigue, anorexia, significant unintentional weight loss (15 kg over two months) and febrile episodes reaching 38.5°C. Notable clinical findings included a perianal abscess. His prior SLE manifestations included non-erosive polyarthritis, serositis (pleurisy and mild pericarditis) and hematologic abnormalities such as hemolytic anemia and lymphopenia. Serologic tests were positive for ANA, anti-SSA, anti-DNA, nucleosome, RNP and histone antibodies. Initial treatment involved corticosteroids (0.5 mg/kg/day) and hydroxychloroquine, which was later discontinued due to cutaneous hypersensitivity.

On current admission, laboratory findings showed pancytopenia: hemoglobin at 5.4 g/dL (normocytic, normochromic), WBC 2,600/mm³ (with 130 neutrophils and 1,040 lymphocytes) and platelet count of 16,000/mm³. CRP was elevated to 232 mg/L and ferritin measured 34,000 ng/mL. Microbiological analysis of the perianal abscess revealed Escherichia coli. Pelvic MRI identified a posterior anal fistula without abscess formation. Peripheral smear demonstrated 55% blasts and bone marrow aspirate confirmed AML with Auer rods and translocation t(8;21).

Initial management included triple antibiotic therapy (imipenem, metronidazole, amikacin), fluconazole (400 mg/day) for antifungal prophylaxis and intravenous methylprednisolone (120 mg/day for 3 days), followed by oral corticosteroids (60 mg/day). The patient was referred to the hematology department, where he received hydration, phenotype and leukocyte-filtered RBC transfusions and induction chemotherapy with favorable clinical and hematological response (Table 1).

Table 1: Paraclinical Data of the Patient

	Data at diagnosis	After treatment	Reference range
HGB (g/dl)	5	14	12.0-13.0 g/dl
MCV (fl)	82	90	80-100 fl
MCHC (g/dl)	36	32	31-36% Hb/cell
WBC (elements/mm3)	2600	9300	4,000-10,000/mm ^{3}
PNN	130	4600	1,500-7,000/mm^3
Lymphocytes (elements/mm3)	1040	4000	1,500-4,000/mm^3
Platelets (elements/mm3)	16000	402000	150.000-400.000/mm^3
ANA (IU/ml)	1:100	NA	< 1:60 IU/ml
DNA Antibodies (IU/ml)	6	NA	<10 IU/ml
C3 (g/l)	NA	NA	0.90-2.10 g/l
C4 (g/l)	NA	NA	0.10-0.40 g/l
CRP (mg/dl)	216	7	<0.3 mg/dl
AST (IU/ml)	20	24	6-25 IU/ml
ALT (IU/ml)	42	12	6-25 IU/ml

HGB: Hemoglobin; WBC: White Blood Cells; MCV: mean corpuscular volume; MCHC: Concentration of corpuscular average hemoglobin; ANA: anti-nuclear antibodies; DNA antibodies: Anti-Acid Deoxyribonucleic Antibodies; C: Complement; CRP: C-reactive protein; AST: aspartate amino transferase; ALT: alanine amino-tranferase; NA": not available.

DISCUSSION

Autoimmune diseases, particularly autoimmune rheumatic diseases, have been reported in patients with myeloid neoplasms, including myelodysplastic syndrome, chronic myelomonocytic leukemia, acute myeloid leukemia and myeloproliferative neoplasms, with a prevalence ranging from 1.5% to 33% [1,2]. The coexistence of systemic lupus erythematosus (SLE) and acute myeloid leukemia (AML) is rare and presents considerable clinical challenges. Apor et al, showed that SLE was associated with an increased risk of leukemia (SIR 2.3, 95% CI 1.9-2.7) [3]. The risk of developing myeloid neoplasms depends on a number of factors, including the chronicity and severity of autoimmune disease, the type and duration of exposure to disease-modifying antirheumatic drugs and genetic predisposition [4]. Some case studies suggest a link with prior exposure to cytotoxic or immunosuppressive drugs [5]. In contrast, Lofstrom et al. did not observe any difference in the frequency of cytotoxic exposure between the case and control cohorts, suggesting that prior exposure to these drugs is unlikely to be a major cause of AML development in SLE patients [6]. Leukopenia was identified as a risk factor for the development of myeloid leukemia and myelodysplastic syndrome was frequently observed. Therefore, bone marrow evaluation should be considered in SLE patients with persistent leukopenia and long-standing anemia[6]. Immunologic dysregulation is a common feature to both AML and SLE. NF-kB is a central mediator in the activation of pro-inflammatory genes and is involved in both AML and SLA [7,8]. Persistent activation of NF-kB

in chronic inflammatory conditions can eventually override inhibitory feedback mechanisms, leading to sustained NF-KB activity[9]. The higher incidence of cancer in patients with chronic inflammation may partly be explained by this constitutive NF-KB activity, which exerts a pro-tumorigenic effect [4]. In addition, no temporal relationship was found between drug exposure and the development of myeloid neoplasia [10]. One study reported the effect of SLE latency on the development of acute myeloid leukemia [11].

The simultaneous presence of these two conditions in the same patient complicates both diagnosis and treatment. The differential diagnosis between an exacerbation of SLE and an initial manifestation of AML can be complex, as both conditions may present with similar hematological abnormalities, such as anemia and leukopenia. In this particular case, the pancytopenia observed on admission, combined with the presence of blasts in the peripheral blood, led to a diagnosis of acute myeloid leukemia. Confirmation by myelogram, revealing Auer bodies and a translocation (8;21), clearly differentiated AML from other hematological complications associated with SLE. The treatment of AML in a patient with SLE requires a carefully balanced, multidisciplinary approach. Corticosteroids, often used to control lupus inflammation, can affect the immune response and complicate the management of leukemia. In this case, treatment included triple antibiotic therapy to control infection, antifungal prophylaxis and corticosteroid therapy for SLE, prior to initiation of AML-specific chemotherapy. This strategy aims to stabilize the patient's condition while controlling both the autoimmune disease and the hematological malignancy. The prognosis of these patients depends on response to treatment of AML and ongoing management of SLE. The presence of the (8;21) translocation is generally associated with a better prognosis in AML, but coexistence with SLE can influence the clinical course. Close follow-up is crucial to monitor potential complications, adjust therapies according to tolerance and response and prevent relapses of either disease. Few similar cases are reported in the literature, which underlines the importance of this case for a better understanding of the interactions between autoimmune diseases and hematological cancers. Further studies are needed to explore the mechanisms underlying this coexistence and to develop optimal treatment protocols.

CONCLUSION

This case illustrates the clinical complexity of the coexistence of SLE and AML, highlighting the associated diagnostic and therapeutic challenges. It also highlights the importance of a multidisciplinary approach and increased vigilance in the follow-up of these patients, in order to maximize the chances of survival and improve quality of life.

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