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Evolving myelodysplastic syndrome in an HIV patient with history of anal cancer and chemotherapy

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Abstract

The incidence of therapy-related myelodysplastic syndromes (t-MDS) has been increasing with the widespread use of highly active antiretroviral therapy (HAART) therapy for HIV and chemotherapy for AIDS-related cancers. The classical dysplastic features in the granulocytes and megakaryocytes may not be easily appreciated. The most reliable distinguishing feature between the hematopoietic dysplasia of t-MDS and that of HIV infection rests on the identification of MDS-related cytogenetic aberrations. Here we report a patient with well-controlled HIV and history of chemotherapy for invasive anal squamous cell carcinoma who developed high-risk t-MDS with complex chromosome abnormalities. Our study emphasizes the importance of diagnosis of MDS in HIV-infected patients, even in the absence of dysplasia, if there are typical cytogenetics changes of MDS. Therefore, the early diagnosis and intervention of t-MDS in HIV-positive patients are critical in the treatment of this aggressiveness disease.

Keywords Therapy-related myelodysplastic syndromes (t-MDS) · Acquired immune deficiency syndrome · HIV · Cancer · Chemotherapy

A 60-year-old white male presented with worsening fatigue for 2 weeks. The patient had a longstanding history of HIV infection and clinical complications of acquired immune deficiency syndrome (AIDS) including cryptococcal meningitis. He was diagnosed with invasive anal squamous cell carcinoma 6 years ago and received mitomycin and radiation therapy. His HIV infection was well controlled by highly active antiretroviral therapy (HAART) treatment. The most recent CD4 T cell count was 299/μL. Peripheral blood smear showed pancytopenia. Complete blood count (CBC): WBC 2.91×10^9/L, neutrophils 34%, lymphocytes 61%, monocytes 4%, eosinophils 1%, basophils 0%, RBC 2.81×10^{12}/L, HGB 7.2 g/dL, HCT 22.5%, MCV 80.1 Fl, and PLT 14×10^9/L. Bone marrow biopsy revealed hypercellular (80% cellularity) marrow with left-shifted granulopoiesis, clusters of blasts, megakaryocytic and erythroid dysplasia (Fig. 1a: blue arrow large cells are blasts, red arrow indicates irregular nuclear dysplastic erythroid precursor; Fig. 1b: green arrow indicates dysplastic megakaryocyte, blue arrow indicates immature mononuclear cells/blasts), and reactive lymphoid aggregates. The immunostaining of CD34 highlighted interstitially increased clusters of blasts (3–4%) (Fig. 1c). Flow cytometry report revealed a 58% maturing granulocytic elements with decreased orthogonal light scatter properties (suggesting hypogranularity) and with an unusual maturation spectrum, and an increased distinct myeloblasts population with immunophenotypical aberrancy (CD34+/CD117+/CD33+/subset CD56+/CD7dim+/HLA-DR+) (Fig. 1d, highlight in red). Cytogenetic analysis found the following complex karyotype of multiples chromosome abnormalities. 43,X,-Y,-5,del(7)(q22q36),inv(12)(p13q15),der(13)del(13)(q14q34)-t(5;13)(p13;q14),-17[5]/43,idem,-inv(12),+der(12)t(12;17)(p12;q21)[5]/43,idem,-inv(12),+der(12)inv(12)(p13q24.1)add(12)(p13)[5] (Fig. 1e).

The patient was diagnosed with therapy-related myelodysplasia (t-MDS) and treatment with azacitidine was initiated. The patient developed progressive pancytopenia and passed away in 3 months. Dysplastic changes to the marrow are a common finding in advanced

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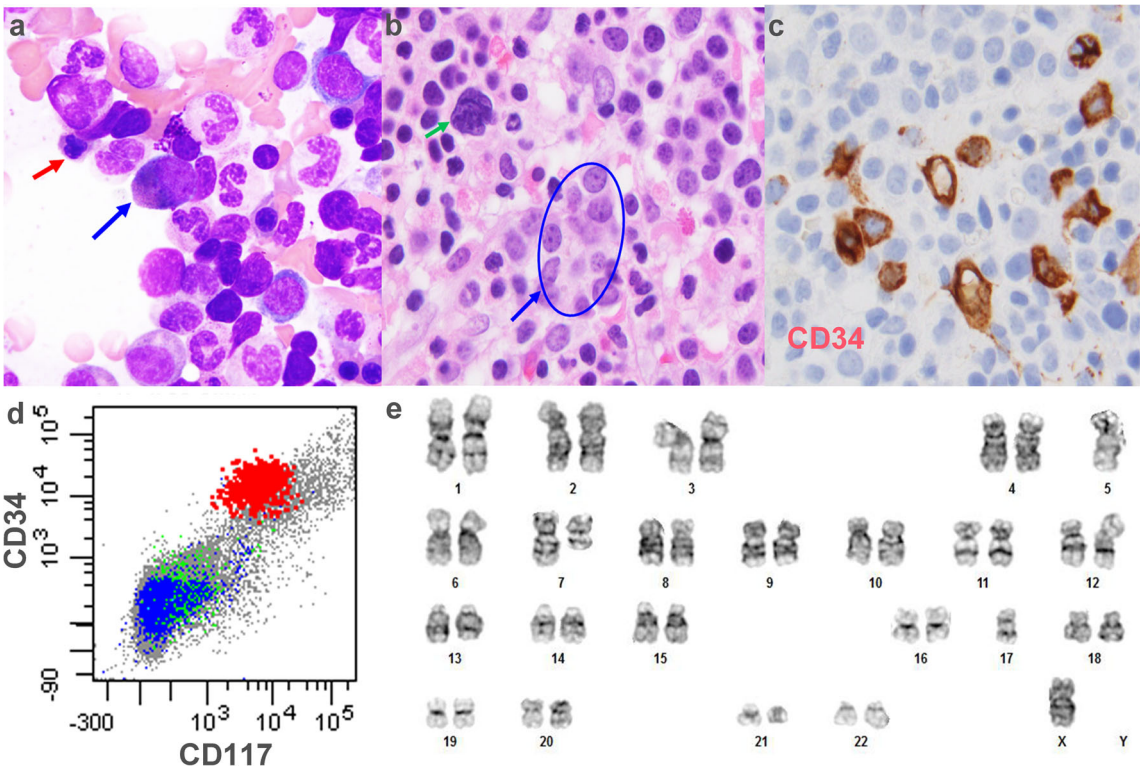

Fig. 1 **a** Morphological, immunophenotypical and cytogentic pathological findings of the case, **b** Bone marrow biopsy, **c** immunostaining of CD34, **d** flow cytometry report, and **e** cytogenetic analysis of a 60-year-old HIV patient

HIV infection on HAART therapy [1]. This case represents a patient with well-controlled HIV and history of chemotherapy who developed high-risk t-MDS with complex chromosome abnormalities. The incidence of t-MDS has been increasing with the widespread use of HAART for HIV and chemotherapy for AIDS-related cancers [2, 3]. The most reliable distinguishing feature between the hematopoietic dysplasia of t-MDS and that of HIV infection rests on the identification of MDS-related cytogenetic aberrations [4, 5]. The early diagnosis and intervention of t-MDS in HIV-positive patients are critical in treatment of this aggressiveness disease.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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