Case Report

Association of Acute Myeloid Leukemia and Systemic Lupus Erythematosus: A Case Report

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Abstract: Systemic lupus erythematosus is an autoimmune inflammatory disease that can affect multiple systems and organs of the body including skin, kidneys, lungs, joints and nervous system. Some previous studies up light that SLE was associated with increased risk of acute leukemia (SIR = 2.3). The incidence of the association of AML and SLE is not known but there are few case reports in literature. The aim of this report was to up light the difficulties which occurred in the diagnosis and the management of acute myeloid leukemia in patient with systemic lupus erythematosus. Patient, 43 years old, diabetic treated with insulin, whose sister is followed for Behcet’s disease, was admitted for AML with trisomy of chromosome 4 and 8 and thrombophlebitis of the superior sagittal sinus. Chest CT-Scan showed minimal bilateral pleural effusion. She was treated according to Morocco National protocol AML-MA-2011. CT-Scan was done at day 12 of chemotherapy and noticed the persistence of the minimal bilateral pleural effusion. At day 19 the patient presented malar rash, and right axillary adenitis. The biopsy of the adenitis showed the presence of LE cells. According to internal physician recommendations we add steroids to the treatment. The cough and fever disappeared. The CT-scan for control was normal after two weeks of steroids. Patient is in complete remission after induction I. After a follow-up of five months, the patient still well, but present severe infections during chemotherapy cycles and a bad tolerance for the treatment. The association SLE-AML is rare. The diagnosis of the association SLE-AML is difficult. It is management is also difficult according to comorbidity, severe infectious because of decline of immunity, and less tolerance to AML chemotherapy.

Keywords: Acute Myeloid Leukemia, Systemic Lupus Erythematosus, Association

1. Introduction

Systemic lupus erythematosus (SLE) is an autoimmune inflammatory disease that can affect multiple systems and organs of the body including skin, kidneys, lungs, joints and nervous system. Some previous studies up light that SLE increase the risk of hematologic malignancies which are definitea heterogeneous group of diseases characterized by the abnormal (malignant) growth and/or accumulation of hematopoietic cells in the blood, bone marrow and/or lymph nodes.

In 2005, a meta-analysis conducted by Zintzaras and al, had shown that there was a moderate risk of lymphoma incidence in patients with SLE with an estimated standardized incidence ratio (SIR) 7-times higher compared with the general population [1]. In 2014, in a meta-analysis, including 401 cases of hematologic malignancies identified in a total cohort of 67,929 individuals with a diagnosis of SLE, Emmanuel Apor and al had shown that SLE was associated with increased SIR of acute leukemia (SIR = 2.3) [2]. The incidence of the...
association of Acute Myeloid Leukemia and SLE is not known but there are few case reports in literature. The first case had been reported by Lee in 1955[3].

This is a report of a case of systemic lupus erythematosus associated with Acute Myeloid Leukemia in an young woman.

2. Case Presentation

Patient, 43 years old, mother of 02 children, diabetic treated with insulin, with a sister followed for Behcet's disease, presented one month before the first consultation, asthenia, cutaneous pallor, rebel's headache, multiple arthralgia, cough and fever. The initial clinical examination had noticed a patient with Performans status (WHO) at 2, febrile at 39°Celsius, blood pressure was at 110/80 mmHg, respiratory rate was at 20 cycles/min. Heart and lung auscultation was normal. No hemorrhagic syndrome, no lymph nodes, no splenomegaly, no hepatomegaly. CBC showed hemoglobin level at 7.8g/dl, white blood cells was at 2430/ml with 54% blasts, platelet was at 56000/ml. The bone marrow aspiration and immunophenotyping concluded to AML 2. The karyotype found trisomy of chromosome 4 and 8. The viral serology were negative. Tuberculosis PCR and Galactomanann antigenemia was negative. Blood cultures were negative. The research for lupus antibodies, nuclear and DNA antibodies were negative. MRI found thrombophlebitis of the superior sagittal sinus (Figure 1). Chest CT-Scan showed minimal bilateral pleural effusion.

Figure 1. Thrombophlebitis of the superior sagittal sinus.

Figure 2. LE Cells.

A multidisciplinary consensual meeting of hematologists, pulmonologists, neurologists decided to treat the patient according to protocol AML-MA-2011, recommend to administrate 0.6 ml of enoxaparin twice a day, to maintain by
The presence of haematological abnormalities, anaemia, leucopenia and to a lesser extent thrombocytopenia are common clinical manifestations of the SLE disease, often independent of disease severity and can be present also in AML.

So it is necessary that SLE patients be subject to regular check-ups for clinical manifestations and laboratory tests, to show if a possible transition had occurred from cytopenias via MDS to AML. Interestingly, leucopenia was the only clinical SLE-associated finding with a significantly elevated OR for leukaemia development.

The frequency of a preceding myelodysplastic phase before leukaemia was at least comparable with the estimated 25% that has been observed in the general leukaemia population [7]. Some previously known associations between rheumatic diseases and leukaemia do exist. In a population-based cohort of Wegener’s granulomatosis, a more than 5-fold increased risk of leukaemia was found [8] [9].

The relation between SLE and leukemia is rather unclear. One could hypothesize that some of the drugs used to treat SLE, such as the alkylator cyclophosphamide among others, can increase the risk of developing myeloid neoplasms such as myelodysplastic syndrome (a pre-leukemic state) and acute myeloid leukemia [10] [11].

So chemotherapeutic drugs, constitute one of the relatively few known aetiological risk factors for leukaemia. Azacytidin is an anti-metabolite that has been used in SLE treatment since the 1960s both for treating disease manifestations and as a glucocorticoid-sparing drug. It could induce defective DNA mismatch repair, possibly promoting survival of cells for a leukemic clone [12]. On the contrary, anti-malarial drugs like chloroquine, are immune modulating drugs often used in patients with SLE that have recently been reported to exert anti-neoplastic properties. They are strongly DNA intercalating, preventing mutations in cells with a high mitotic rate and improving cellular mechanisms of DNA repair after the damage was caused by alkylating therapy [13]. This effect of anti-malarial drugs can explain why AML are rare in SLE patients.

The therapeutic management of AML in SLE is difficult because of comorbidity (kidneys, lungs, nervous system diseases), severe infectious because of decline of immunity, and less tolerance to AML chemotherapy. In fact febrile neutropenia is the most frequent and important complications of chemotherapy during the management of acute myeloid leukemia. It is generally the first factor of mortality. It incidence had been estimated between 70–100% during the neutropenic phase after intensive chemotherapy. Immune deficiency developed during malignancy and chemotherapy is
the first factor which explain febrile neutropenia in acute myeloid leukemia patient. Because of this immune deficiency serious infection will manifest in neutropenic patient with minimal symptoms. In AML patient with SLE this immune deficiency is more important and patient present graveous infections during neutropenia. It what was noticed also when managed this patient which present several infection during a different phase of chemotherapy.

The median survival time after the diagnosis of AML in SLE patients is 7 months (2-18 months) [14] [15]. In our case, after a following-up of five months, the patient still well, but present sevver infections during chemotherapy cycles and a bad tolerance for the treatment.

4. Conclusion

The association SLE-AML is rare. The presence of haematological abnormalities, are common clinical manifestations of the SLE disease, often independent of disease severity and can be present also in AML. So the diagnosis of the association SLE-AML is difficult. It is management is also difficult according to comorbiditry, sevver infectious because of decline of immunity, and less tolerance to AML chemotherapy.

Abbreviations

Systemic lupus erythematosus (SLE); Acute Myeloid Leukemia (AML).

Authors’ Contributions

RM and MC prepared the draft and BH, ML, BO, and AQ all reviewed and contributed to the final manuscript.

Conflict of Interest

The authors declare that they have no competing interests.

Consent for Publication

Written consent to publish this report was obtained from the patient.

References

[1] Zintzaras E, Voulgarelis M, Moutsopoulos HM. The risk of lymphoma development in autoimmune diseases: a meta-analysis. Arch Intern Med 2005;165:2337-44.

[2] Emmanuel Apor, Jennifer O’Brien, Merin Stephen, Jorge J. Castillo d. Systemic lupus erythematosus is associated with increased incidence of hematologic malignancies: A meta-analysis of prospective cohort studies. Leukemia Research 38 (2014) 1067–1071.

[3] Lee SL. Clinical experience with the LE cell test. J Mount Sinai Hosp NY 1955;22:74–8.

[4] Mary Lu, Sasha Bernatsky, Rosalind Ramsey-Goldman, Michelle Petri, Susan Manzi, Murray B and al. Non-Lymphoma Hematological Malignancies in Systemic Lupus Erythematosus. Oncology. 2013; 85(4).

[5] Kang KY, Kim HO, Yoon HS, et al. Incidence of cancer among female patients with systemic lupus erythematosus in Korea. Clin Rheumatol 2010;29: 381–8.

[6] Bjorn Lofstrom, Carin Backlin, Christer Sundstrom, Eva Hellstrom-Lindberg, Anders Ekbom, Ingrid E. Lundberg. Myeloid leukaemia in systemic lupus erythematosus–a nested case–control study based on Swedish registers. Rheumatology 2009;48:1222–1226.

[7] Julisson G, Antunovic P, Derolf A et al. Real world data on decision to treat and outcomes from the Swedish acute Leukemia Registry. 2009 113: 4179-4187.

[8] Knight A, Askling J, Ekbom A. Cancer incidence in a population-based cohort of patients with Wegener’s Granulomatosis. Int J Cancer 2002;100:82–85.

[9] Knight A, Askling J, Granath F, Speren P, Ekbom A. Urinary bladder cancer in Wegener’s granulomatosis: risks and relation to cyclophosphamide. Ann Rheum Dis 2004;63:1307–11.

[10] Bhatia S. Therapy-related myelodysplasia and acute myeloid leukemia. Semin Oncol 2013;40:666–75.

[11] Bernatsky S, Joseph L, Boivin JF, et al. The relationship between cancer and medication exposures in systemic lupus erythematosus: a case-cohort study. Ann Rheum Dis 2008;67:74–9.

[12] Leone G, Pagano L, Ben-Yehuda D, Voso MT. Therapy-related leukaemia and myelodysplasia: susceptibility and incidence. Haematologica 2007;92:1389–98.

[13] Ruiz-Irastorza G, Ugarte A, Egurbide MV et al. Antimalarials may influence the risk of malignancy in systemic lupus erythematosus. Ann Rheum Dis 2007;66:815–7.

[14] Kwong YL, Au WY, Liang RHS. Acute myeloid leukemia after azathioprine treatment for autoimmune diseases: association with -7/-7q-. Cancer Genet Cytogenet 1998;103:94–7.

[15] Leone G, Pagano L, Ben-Yehuda D, Voso MT. Therapy-related leukaemia and myelodysplasia: susceptibility and incidence. Haematologica 2007;92:1389–98.