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Case Report

Pediatric Lupus and Congenital HIV, A Rare Association with Difficult Therapeutic Management: A Case Report from Burkina Faso

Wendyam Nadège Yameogo[1], Ismael Ayouba Tinni[1]*, Wendlassida Stéphanie Joelle Zabsonre/Tiendrebeogo[1,2], Fulgence Kabore[1,2], Marcellin Bonkoungou[1] and Dieu-Donné Ouedraogo[1,2]

[1]Rheumatology Department, CHU Bogodogo, Burkina Faso

[2]Joseph KI-ZERBO University, Burkina Faso

***Corresponding Author:** Ismael Ayouba Tinni, Rheumatology Department, CHU Bogodogo, Burkina Faso.

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Abstract

Introduction: The coexistence of congenital acquired immunodeficiency syndrome with systemic lupus erythematosus (SLE) is rare, especially in the pediatric population. We report a pediatric case, the first case reported in Burkina Faso.

Observation: This was an 11-year-old female patient, seropositive to human immunodeficiency virus (HIV) type 1, followed since the age of 18 months with an undetectable viral load, on Abacavir-Dolegravir-Ritonavir/day and cotrimoxazole. She was admitted for chronic inflammatory polyarthralgia associated with exertional dyspnea, of chronic evolution in a context of altered general condition. The immunological work-up noted: positive antinuclear = 1280 IU/ml. Anti DNA antibody = 380 IU/ml, anti Sm antibody = 33U/ml, anti-histone antibody negative. The diagnosis of systemic lupus erythematosus in HIV was retained.

Conclusion: SLE and HIV are two pathologies with some clinical similarities, but the therapeutic objectives and the medication are divergent to the point where the association of the two in the same patient complicates the management.

Keywords : SLE; Child; HIV; Burkina Faso

Introduction

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease of unknown etiology, characterized by the production of multiple autoantibodies, the most characteristic of which are directed against certain nuclear components [1]. It mainly affects women in adulthood and is rare in children, with only 10-17% of cases diagnosed before the age of 16 [1,2]. Some of the clinical manifestations of this disease are similar to those found in the acquired immunodeficiency syndrome (AIDS), the coexistence of these two diseases is rarely reported in the world. However, with the reconstitution of the immune system in the era of highly active antiretroviral therapy (HAART), an increasing number of cases of SLE are being reported, some of which are even attributed to the inflammatory syndrome of immune reconstitution [3,4]. Until 2020, publications on the coexistence of SLE and human immunodeficiency virus (HIV)-1 infection were single case reports, most involving SLE occurring in children in the context of congenital HIV-1 infection [4-6]. We report a case of recurrent pediatric systemic lupus erythematosus in a congenital HIV-1 background, the first reported case of co-existing SLE and HIV infection in a patient in Burkina Faso.

Observation

Eleven-year-old female patient, first twin, primary school Class 6 student, living in an urban area, HIV positive type 1 regularly followed since the age of 18 months with an undetectable viral load, on tenofovir-lamivudine, dolutegravir one tablet per day and cotrimoxazole 480 mg one tablet per day. As family history, the mother and the second twin are also HIV positive and on treatment. She was admitted to the rheumatology department for chronic inflammatory polyarthralgia associated with exertional dyspnea of chronic evolution in a context of altered general condition. The interrogation noted a notion of long-term fever. There was no family history of rheumatological disorders. The examination revealed a general condition of WHO stage 3, clear consciousness, fever at 37.8 degrees Celsius, eight painful joints, poly-seritis (pulmonary, cardiac), smooth, painless, firm hepatomegaly. Non-specific dermatological lesions of LES were also noted due to alopecia, melanoderma, curly, red and brittle hair evoking a silky tricopathy. The biological work-up revealed normocytic normochromic anemia at 9.9 g/dl, 24-hour proteinuria: 885 mg/24h. Immunological work-up: antinuclear positive = 1280 IU/ml. Anti DNA negative = 380 IU/ml, anti Sm (SmD3) = 33U/ml, anti-histone negative. Chest X-ray showed pleurisy with moderate pleural effusion. Cardiac Doppler ultrasound noted a circumferential pericardial detachment with moderate fluid effusion. Abdominal ultrasound noted homogeneous hepatomegaly, bilateral renal pain with no intraperitoneal fluid effusion. The diagnosis of systemic lupus erythematosus in HIV was retained. A treatment based on corticoid dose/weight and hydroxychloroquine 200 mg/day was associated with his antiretroviral tritherapy made of abacavir, dolutegravir, ritonavir and cotimoxazole 480 mg/day.

The patient was re-hospitalized 04 months later with dyspnea without fever. Physical examinations and imaging revealed a bilateral pleural effusion of great abundance on the left with a biological inflammatory syndrome. Cytobacteriological examinations of the pleural puncture fluid did not isolate any germ. Azathioprine at a dose of 50 mg/day was combined with the previous treatment without success because after 03 months the patient was readmitted to the department in a similar picture to the previous one, death had occurred a few days later in a picture of acute respiratory distress, with severe renal failure and OAP.

Discussion

The coexistence of human immunodeficiency virus (HIV) infection and systemic lupus erythematosus (SLE) is rarely observed since SLE is a CD4+ T-cell-mediated process and HIV-1 targets CD4+ T cells, thus causing their depletion and theoretically reducing the risk of SLE [4]. Although rare, concomitant cases of SLE and human immunodeficiency virus (HIV-1) infection have been reported since 1988 [7]. Indeed, nine cases of association of pediatric systemic lupus erythematosus and congenital HIV have been reported in the literature to our knowledge. Among these nine cases, there were seven cases of lupus nephropathy without any other clinical manifestation, one case of lupus nephropathy and secondary vasculitis, and one case of joint manifestation [4-9]. In our patient, pulmonary, rheumatological and dermatological involvement were the circumstances of discovery, whereas most studies noted a high frequency of renal involvement, which is a formidable cause of functional disability and death, in case of early onset of SLE.

On the familial level, in the twin and the mother of our patient no manifestation of SLE had been observed, apart from those of HIV-1. The frequency of familial lupus varies from 4 to 12% according to the series, which underlines the role of innate factors in the occurrence of SLE. The concordance rate in twins (proportion of second affected twin when the first is ill) is a strong argument in favor of a genetic component. This rate varies from 24% to 56% in monozygotic twins, whereas it is only 2% to 4% in dizygotic twins [10].

In fact, these 2 conditions share multiple overlapping clinical features, including hematologic, neurologic, renal, and other abnormalities. They can sometimes be indistinguishable. Nevertheless, the overproduction of ANA and anti-dsDNA antibodies is a feature of SLE. ANAs have diagnostic, pathological and prognostic significance during the course of lupus disease. However, HIV-infected individuals may also produce autoantibodies, such as ANA and anti-cardiolipin antibodies, in small amounts [11] causing diagnostic difficulties. It should also be noted that recent studies have revealed that polyreactivity or autoreactivity is one of the main characteristics of anti-HIV antibodies (autoreactive lymphocytes), especially antibodies

with broad and potent neutralizing abilities. These may play an etiological role in autoimmune diseases [12]. In addition, patients with SLE may also produce antibodies that cross-react with HIV antigens, leading to false-positive tests for HIV infection [13], viral load and CD4+ counts should be determined in these situations. Although the association of SLE and HIV is rare, the two conditions are not mutually exclusive as previously thought.

The management of SLE in the context of HIV is challenging as most treatments cause immunosuppression and may lead to a higher risk of opportunistic infections and disease progression in an already vulnerable population [4]. Indeed, an increase in HIV viral load after control of SLE by immunosuppressive therapy has been reported in several patients with both diseases. However, improvement or stabilization of SLE and HIV-1 has been observed in several studies [14,15]. Several authors [3-9] corroborate that in SLE patients with confirmed HIV infection; antiretroviral therapy should be considered before immunosuppressive therapy. In addition, monitoring of CD4+ T-cell counts is strongly recommended to determine the appropriate immunosuppressive dose.

Conclusion

There are challenges in the therapeutic management of this combination of pediatric SLE and HIV, as there are currently no specific recommendations for children for either lupus or its combination with HIV. This situation is very embarrassing for the rheumatologist, especially in Africa, given the significant prevalence of congenital HIV.

Conflict of Interest

None.

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