

A Systemic Lupus Erythematosus Revealing by Lymphadenopathies: About 3 Cases

Ladji Mohamed Diaby^{1*}, Abass Sanogo², Abdrahamane Salia Maiga¹, Salif Sow², Youssouf Camara², Lassina Diallo², Drissa Kaloga Bagayoko¹, Aboubacar Sidiki Thissé Kane²

¹Military Hospital of Kati-MHK, Koulikoro, MALI

²Military Hospital of Bamako-MHB, BAMAKO, MALI

*Corresponding author

Ladji Mohamed Diaby

Article History

Received: 11.02.2018

Accepted: 18.03.2018

Published: 30.03.2018

DOI:

10.36347/sjmcr.2018.v06i03.012



Abstract: Systemic lupus erythematosus is a non-specific organ autoimmune disease characterized by clinical polymorphism and positive antinuclear antibodies. The aim of this study was to evaluate the presence of lymphadenopathies during lupus disease. It is a retrospective study of six (06) patients who are married, Malians and suffering from lupus disease. The study was carried out at the service of internal medicine of Military Hospital of Kati over a two (02) year period (from December 2015 to January 2017). The only criteria for inclusion were the presence of one or several lymphadenopathies without considering the size. Lymphadenopathies were found in three lupus patients among six (50% of patients); the mean age was 26, 33 years. Peripheral lymphadenopathies involved cervical area in one case (16, 66%), inguinal area in two cases (33, 34%). In all patients, all of the following exams were positive: anti-native DNA antibodies, anti-Sm antibodies, anti-Ro/SS-A antibodies and/or anti-La/SS-B and hypocomplementaemia. Development under Corticotherapy appeared to have a favorable effect with adenopathy regression in five patients. Only one case didn't receive methotrexate and its lymphadenopathies regression were noticeable. Lymphadenopathies generally occur during lupus outbreak. However, an investigation for an etiology is necessary in order to eliminate possible infectious cause especially tuberculosis.

Keywords: Lymphadenopathy, Systemic lupus erythematosus, clinical case, Military hospital.

INTRODUCTION

Systemic lupus erythematosus is a non-specific organ autoimmune disease characterized by a clinical polymorphism and positive antinuclear antibodies [1]. Hematologic effects are frequent; they are essentially manifested by a blood cytopenia. Lymphadenopathies are less frequent, observed in 18-42% of cases; and during lupus development, the cervical lymphadenopathy form is the most frequent (30-70% of cases). In case of histological examination, we observe benign follicular hyperplasia [2]. The aim of this study was to evaluate the presence of lymphadenopathies during lupus disease.

PATIENTS AND METHODS

It is a retrospective study of six (06) patients who are married, Malians and suffering from lupus disease. The study was carried out at the service of internal medicine of Military Hospital of Kati over a two (02) year period (from December 2015 to January 2017). The diagnosis of systemic lupus erythematosus was confirmed according to American College of Rheumatology (ACR) 1990 criteria. The only criteria

for inclusion were the presence of one or several lymphadenopathies without considering the size. For statistical study, we used chi²-test for qualitative variables and Student test for quantitative variables.

OBSERVATIONS

Lymphadenopathies were found in three lupus patients among six (50% of patients); it was about three women (100%) whose mean age was 26, 33 years (extreme: 18-35 years). All patients presented peripheral lymphadenopathies associated with deep lymphadenopathies. Peripheral lymphadenopathies involved cervical area in one case (16, 66%), inguinal area in two cases (33,34%).

Biological investigation: Generalized elevation of sedimentation rate was noticed in all patients; C-reactive protein (CRP) value was remained normal in all women, except for the tuberculosis patient who had an elevated CRP rate. A polyclonal Hyperimmunoglobulinemia composed of IgG and a leukopenia associated with anemia were detected in all patients.

In all patients, all of the following exams were positive: anti-native DNA antibodies, anti-Sm antibodies, anti-Ro/SS-A antibodies and anti-La/SS-B and hypocomplementaemia.

A lymph node biopsy concluded in non-specific lymphadenitis (one case), histiocytic necrotizing lymphadenitis of Kikuchi Fujimoto (one case) and lymph node tuberculosis (one case).

The lymph node tuberculosis case regressed with tuberculosis treatment. In all other cases, corticotherapy dose was variable and depended on systemic effects of the lupus disease.

Development under corticotherapy (prednisolone 10mg a day), hydroxychloroquine (400mg a day), and methotrexate (25mg a day) were favorable with adenopathy regression in five patients. The lymph node tuberculosis case didn't receive methotrexate and its lymphadenopathies regression was noticeable.



Fig-1: cervical lymphadenopathies

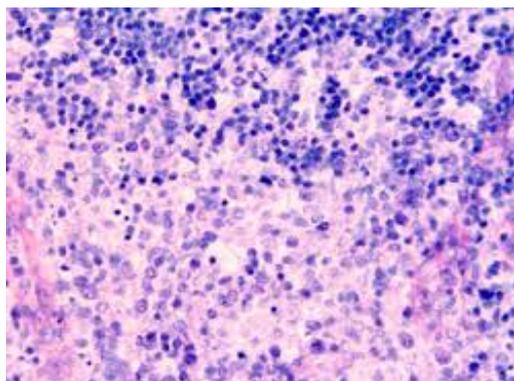


Fig-2: Histiocytic necrotizing lymphadenitis

DISCUSSION

At some point in the disease course, lymphadenopathies are found in 19 to 69% of patients. There are lymphadenopathies of an inflammatory type; it could be localized cervical lymphadenopathies (16%) for Harvey [2] or generalized (34%) [3]. They frequently occur in children and black people. For certain authors, their presence is a good indicator of

clinical signs improvement of the disease [4]. All lymph nodes could be involved: superficial, but also deep and mediastinal lymph nodes [5], and realizing an aspect of bilateral hilar lymphadenopathy [6]; despite the corticotherapy, their extension could lead to performing biopsy which generally shows a hyper-immune lymph node with sometimes hematoxylin bodies and central necrosis. The aspect realized is close or at least similar to histiocytic necrotizing lymphadenitis of Kikuchi Fujimoto [7].

Some lymphographies performed during polyadenopathy could realize an aspect of pseudolymphoma which regresses under corticotherapy [8]. They may be the source of superior vena cava syndrome [9]. Lymph node sarcomas and malignant blood diseases will be considered as part of morbid associations. In addition, some Sternberg cells have been observed in lupus lymphadenopathy, without any Hodgkin disease which could be also associated with lupus erythematosus disease. Finally, some infection of HIV-1 could conceal a lupus erythematosus disease [10].

CONCLUSION

Lupus is a frequent connective tissue disease in genital activity woman; which precise etiology is unknown. Lymphadenopathies are less frequent during lupus disease. However, an investigation is very important in order to discard an infectious source especially tuberculous.

REFERENCES

1. Abeles M, Urman JD, Rothfield NF. Aseptic necrosis of bone in systemic lupus erythematosus: relationship to corticosteroid therapy. *Archives of Internal Medicine*. 1978 May 1;138(5):750-4.
2. Harvey AM et coll, systemic lupus erythematosus. *Rev of literature and clinical analysis of 138 cases*. *Medicine*. 1954, 33: 291-437.
3. Fox RA, Rosahn PD. Lymph nodes in disseminated lupus erythematosus. *Am J Pathol*, 1943. 19 : 73-99.
4. Shapira Y, Weinberger A, Wysesbeek AJ. Lymphadenopathy in systemic lupus erythematosus. Prevalence and relation to disease manifestations. *Clinical rheumatology*. 1996 Jul 1;15(4):335-8.
5. Pequignot M. et coll. Adenopathies mediastinales resolutives au cours d'une maladie lupique révélées par des thromboses veineuses répétées. *Ann Med Int*, 1972, 123 : 961-966.
6. Kassan SS. et coll progressive hilar and mediastinal lymphadenopathy in systemic lupus erythematosus on corticosteroid therapy. *N Engl J Med*, 1976, 294: 1382-1383.
7. Dorfman RF, Beary GV. Kikuchi's histiocytic necrotizing lymphadenitis: an analysis of 108 cases with emphasis on differential diagnosis. *Sem Diag Pathol*, 1988, 5: 329-345.

8. 1277. Herbeuval R, thibaut G, guerci O, Et Coll. Adenopathies profondes et lupus. *Ann Med Interne*, 1977, 128 : 43-48.
9. Van den Brink H, Vroom TM, Van de Laar MA, Van Soesbergen RM, Van der Korst JK. Superior vena cava syndrome caused by systemic lupus erythematosus in a patient with longstanding rheumatoid arthritis. *The Journal of rheumatology*. 1990 Feb;17(2):240-3.
10. De Clerck LS, Couttenye MM, De Broe ME, Stevens WJ. Acquired immunodeficiency syndrome mimicking Sjögren's syndrome and systemic lupus erythematosus. *Arthritis & Rheumatology*. 1988 Feb 1;31(2):272-5.