

Neuropsychiatric manifestations of Systemic Lupus Erythematosus

Melisa Uruci*, Dorina Ruci, Nerenxa Ajasllari

University Medical Center of Tirana "Mother Teresa"

*Corresponding author: Melisa Uruci, University Medical Center of Tirana "Mother Teresa"

Received date: 13 July, 2022 |

Accepted date: 24 July, 2022 |

Published date: 27 July, 2022

Citation: Uruci M, Ruci D, Ajasllari N. (2022) Neuropsychiatric manifestations of Systemic Lupus Erythematosus. J Case Rep Med Hist 2(3): doi <https://doi.org/10.54289/JCRMH2200111>

Copyright: © 2022 Uruci M, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Key Words: Systemic Lupus Erythematosus; Neuropsychiatric Disorder; Glomerulonephritis Class IV; Pulse Therapy with Methylprednisolone; Autoantibodies; Anti SSA; Ant- Rib-P

Abbreviations: Rib-P: Ribosomal

Introduction

Neuropsychiatric manifestations of Systemic Lupus Erythematosus display a wide clinical spectrum with cerebrovascular accidents, seizures, cognitive disorders, demyelinating syndromes, psychosis, optic myelitis, myelitis. The passage of antibodies through the blood-brain barrier to the central nervous system, neuroinflammation, and cerebral ischemia are the main mechanisms of neuropsychiatric involvement in these patients [1]. The elevated activity of Lupus Erythematosus disease or previous damaging events of the brain (presence of positive antiphospholipid antibodies) may also be associated with neuropsychiatric lupus. But we must also take into consideration the impact of high-dose cortisone therapies (influenced by high proteinuria and hypoalbuminemia) that can cause the start of some psychiatric symptoms [2]. In this regard, some authors support the theory of increasing the dose and wait for the clinical response in the coming days, while others refer to a rapid tapering and removal of the drug that caused the effect [3].

Case Presentation

Patient with initials A.H, 33 years old diagnosed with Systemic Lupus Erythematosus in November 2018. The

patient was on treatment with Plaquenil 400 mg per day and prednisone 5 mg per day presenting in good condition but with manifestation of two episodes of anxiety disorders and depression due to difficult socioeconomic conditions. The patient comes to the hospital in March 2021 with malar rash, photosensitivity, oral ulcers, proteinuria 3800 mg/24 h. The patient performed a renal biopsy resulting in Glomerulonephritis class IV. The following results are observed in laboratory examinations: C3 70 mg/dL, C4 8.0 mg/dL (low), Anti Sm 20 U/mL (High), Anti SSA 200 U/mL (high), Anti SSB 10 U/mL (in normal range), FR neg, Anti CCP neg, ENA-screen 2,3 index/ml (High), ANA 1: 320, Anti Cardiolipin IgG, IgM neg, Lupus Anticoagulant neg, Anti β 2 glycoprotein 1 IgM, IgG neg. Anti ds DNA 88.3, Serum albumin 2.7 g/dl, Total protein 3.2 g / dl.

The patient started pulse therapy with Methylprednisolone 1000 mg/day for three days. In the afternoon of the second day of pulse therapy the patient began to have hyperactivity, agitation, ideas of persecution and auditory and visual hallucinations. The patient was diagnosed by a psychiatrist with Acute Psychosis, who initiated treatment with antipsychotics and antidepressants. The patient immediately underwent magnetic resonance imaging of the head resulting negative for vascular and parenchymal abnormalities. The



patient, after finishing pulse therapy, started therapy with MMF 2 g/day, Prednisone 0.5 mg/kg of body weight and ACE inhibitors and her condition started to improve. In the follow-up consult she made after 6 months, the patient's white and red cells in the laboratory tests were in the normal range, proteinuria was reduced to 200 mg/24 h and also the inflammatory tests resulted normal.

Discussion and Conclusions

Under these circumstances, we could suspect that the patient, having a previous history of anxiety disorders and depression, being also induced from the onset of high doses of cortisone had precipitated into acute psychosis. We can also identify the association of present autoantibodies in hyperinflammatory syndromes that cause inflammation of the central nervous system such as the presence of SSA anti Sm and Anti ds DNA in this patient [4]. We could also investigate the presence of anti-ribosomal (anti -Rib-P) antibodies which in this case would result positive, (they are one of the antibodies detected in the cerebrospinal fluid or in the serum of a patient with Lupus exhibiting neuropsychiatric disorders), study conducted since 1999 by the American College of

Rheumatology [5], but this test is not performed in our country.

Reference:

1. Kivity S, Agmon-Levin N, Zandman-Goddard G, Chapman J, Shoenfeld Y. (2015) Neuropsychiatric lupus: a mosaic of clinical presentations. 13: 43.
2. Patten SB, Neutel I. (2000) Corticosteroid-induced adverse psychiatric effects. Incidence, diagnosis and management. *Drug Saf.* 22(2): 111-122.
3. Wysenbeek AJ, Leibovici L, Zoldan J. (1990) Acute central nervous system complications after pulse steroid therapy in patients with SLE. *J Rheumatol.* 17: 1695-1696.
4. (2009) Immunological and histochemical analyses of cerebrospinal fluid and peripheral blood from patients with neurological and psychiatric disorders *Acta Neuropsychiatr.* 2: 51-57.
5. Yoshio T, Hirata D, Onda K, Nara H, Minota S. Antiribosomal P protein antibodies in cerebrospinal fluid are associated with neuropsychiatric systemic lupus erythematosus. *J Rheumatol.* 32(1): 34-39.