

Acute Axonal Sensorimotor Polyneuropathy in Systemic Lupus Erythematosus (Sle) Hemodialysed Patient

Brătescu Lavinia-Olțita¹, Gemene Marta-Emanuela¹, Moga Cristina Gabriela¹, Zugravu Adrian-Dorin^{2,3} and Copăceanu Diana¹

¹Diaverum Morarilor Nephrology and Dialysis Medical Centre, Bucharest, Romania.

²Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania.

³Carol Davila" Nephrology Hospital, Bucharest, Romania.

*Correspondence:

Brătescu Lavinia-Olțita, Consultant in Nephrology Diaverum Morarilor Nephrology and Dialysis Medical Centre, Bucharest, Romania, Tel: +40-721242126; E-mail: lavinia_bratescu@yahoo.com.

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ABSTRACT

There was described the case of acute axonal sensorimotor polyneuropathy in a chronic hemodialysed (HD) patient with systemic lupus erythematosus (SLE). The signs and symptoms and also, the immunological laboratory criteria were initially interpreted only as a spurt of SLE activity and treated accordingly. Lack of response to treatment has led to additional neurological and imaging investigations, who supported the diagnosis of Acute axonal sensorimotor polyneuropathy.

As a treatment, 5 plasmapheresis sessions were conducted followed by quarterly intravenous administration of 2g/kg of body weight Immunoglobulin (5 cures), with improved evolution.

Sustaining of SLE activity is not common in chronic hemodialysed patient. Usually, after start dialysis autoimmune systemic disease shows no activity. On the other hand, the causal relationship between acute axonal sensorimotor polyneuropathy and SLE, has not been definitely established.

Keywords

Hemodialysis, Polyneuropathy, Plasmapheresis, Systemic lupus erythematosus.

Introduction

SLE is most common autoimmune disease in women between 20 and 40 years. Patients present with variable clinical features ranging from mild joint and skin involvement to life-threatening renal, hematologic, or central nervous system involvement [1]. The heterogeneity of signs and symptoms at the first presentation of the patient in the clinic, making the diagnosis difficult. The same thing is also observed in the case of accruals. Serological findings are important in suggesting the possibility of SLE, with some antibodies (eg, anti-double-stranded DNA [dsDNA] and anti-Smith [Sm] highly associated with this condition [1]. 10 to 30% of patients with proliferative lupus nephritis progress to end stage renal disease (ESRD) and needs dialysis. SLE activity is

much attenuated after dialysis started.

Acute motor and sensory axonal neuropathy (AMSAN) is a described subtype of Guillain-Barré syndrome (GBS) characterized by acute onset of distal weakness, loss of deep tendon reflexes and sensory symptoms [2]. Electrophysiological studies show mildly reduced nerve conduction velocities combined with a marked reduction of muscle action and sensory nerve action potentials [2].

Acute axonal sensorimotor polyneuropathy is not common associated with SLE, even when SLE is active.

Materials and Methods

A 48-year-old women, who presents with fever, fatigue, arthralgia, malar rash, leukopenia, severe anemia and low impaired renal function (seric creatinine=1,56mg/dl) was SLE diagnosed in 2002 and initially treated with Medrol and Hydroxychloroquine

(Plaquenil). In 2004 and 2007 patient had hip arthroplasty for aseptic necrosis of the bilateral femoral neck. HD was starting in 2007 due to spurt severe lupus nephritis, intolerant to treatment with Cyclophosphamide. The patient associated also antiphospholipidic syndrome (deep left leg thrombosis and left-cilioretinal artery) and secondary hypertension. The monoclonal gammopathy in the context of progressive and persistent hypercalcemia (multiple myeloma was denied) was associated in 2014.

In 2016, the patient presented to the nephrology clinic with the extreme asthenia, important pain and muscle weakness, lower and upper important bilateral limbs motor deficit, a frigore right peripheral facial paresis, preceded by mild enteritis, symptomatically treated and interpreted as viral. Associated immunological laboratory tests led to initial interpretation as a spurt of SLE activity (IgG, C3, Antibodies dsDNA positive) and treated accordingly with mycophenolate 1g/day and methylprednisolone without improved evolution.

The patient associated loss of deep tendon reflexes, without ataxy or stiff neck. Acute axonal sensorimotor polyneuropathy diagnosis was sustained in the Neurology Clinic by: ENG (electronystagmography)/EMG (electromyography) - acute demyelinating polyneuropathy, motor amplitudes reduced by driving blocks distal - and albuminocytologic dissociation of the cerebrospinal fluid. No space-occupying processes by native brain CT have been highlighted. A right femoral central venous catheter vein was inserted and 5 plasmapheresis sessions were conducted followed by quarterly intravenous administration of 2g/kg of body weight Immunoglobulin (5 cures), with improved clinical and paraclinical evolution.

Discussions and Conclusions

The association of presented signs and symptoms with the laboratory immunological tests in SLE patient could initially be interpreted as a spurt of SLE activity, even in a chronic hemodialysed patient. Impaired neurological symptoms under treatment and the observation that they were preceded by enteritis, led to the patient's guidance to the neurology clinic.

AMSAN, axonal subtypes of GBS cases, preceded by *Campylobacter jejuni* enteritis are reported in the literature. [3]. Cytomegalovirus (CMV), Epstein-Barr virus (EBV) and *Haemophilus influenzae* (*H. influenzae*) and Hepatitis A infection could led also to AMSAN. The exact pathogenesis by which these cause the disease is not clear [3-6].

Acute axonal sensorimotor polyneuropathy diagnosis was more difficult as it was associated with increased SLE activity. The first particularity of the case is the sustaining of SLE activity in HD patient. Usually, after start dialysis autoimmune systemic disease shows no activity.

The second feature of the case is the association of acute axonal sensorimotor polyneuropathy with SLE, whose causal relationship has not been established. The distinction between spurt of SLE activity and acute axonal sensorimotor polyneuropathy is very important for therapy management.

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