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PARKINSONISM CAN BE THE FIRST MANIFESTATION OF LUPUS IN AN OLDER ADULT

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PARKİNSONİZM, GERİATRİK POPÜLASYONDA LUPUS'UN İLK BELİRTİSİ OLABİLİR

Geç başlangıçlı lupus tanısı sinsi başlangıç, düşük prevalans ve immünolojik testlerin başlangıçtaki olumsuzluğu ve SLE kriterlerinin olmaması nedeniyle erken başlangıçtan daha zor olabilir ve gözden kaçabilir.Öte yandan Parkinson hastalığı yaşlı erişkinlerde daha yaygındır, bu da bu popülasyonda lupus teşhisini zorlaştırabilir. Oldukça nadir olsa bile, parkinsonizm yaşlı bir yetişkinde SLE'ye bağlı olabilir. Bu olgu sunumunda, parkinsonizmin SLE'nin ilk belirtisi olabileceği ve geriatrik hasta grubunda steroid tedavisi ile tamamen iyileşebildiği belirtilmektedir.

Anahtar Kelimeler: Parkinsonizm, lupus, yaşlı erişkin

PARKINSONISM CAN BE THE FIRST MANIFESTATION OF LUPUS IN AN OLDER ADULT

The diagnosis of late onset lupus may be more difficult than that of early-onset, and may be overlooked because of insidious onset, low prevalence, and the initial negativity of immunologic tests and the lack of SLE criteria. On the other hand, PD is more common in older adults, which can also make the diagnosis of lupus difficult in this population. Even if it is extremely rare, parkinsonism may be due to SLE in an older adult. In this report, it is presented that parkinsonism can be the first manifestation of SLE, and completely cured by steroid therapy in an older adult.

Keywords: Parkinsonism, lupus, older adult

INTRODUCTION

Parkinsonian signs are common among older adults with prevalence estimates that range from 15 to 40%, and show at least two thirds of patients to be older than 70 years (1). Although Parkinson Disease (PD) is the most common cause of parkinsonism, accounting for approximately 70% of cases, parkinsonism can arise from several causes, including vitamin B12 deficiency, infections, toxins, trauma, but those with the exception of drug-induced parkinsonism are much rarer than PD (1). Because it can lead to gait and balance problems, falls, functional disability, and mortality, early detection and treatment of parkinsonism is quite important (1,2).

Systemic lupus erythematosus (SLE) is a heterogeneous autoimmune disease with highly variable clinical manifestations. SLE often affects women of child-bearing age, but almost 10-20% of cases occur in older adults (3). The changes in hormonal and cellular immunity due to aging may tend to develop SLE in older adults. Many studies suggest that the clinical and serological features of late-onset lupus differ from those of early-onset lupus. For example, neuropsychiatric symptoms such as psychoses, seizures, cognitive dysfunction, neuropathy, headache are more common in older than in younger adults (3,4). However, parkinsonism associated with SLE is quite rare in the literature and all reported cases are young adults (5,6). In this report, it is presented that parkinsonism can be the first manifestation of SLE in an older adult.

CASE REPORT

An 80-year-old woman was brought to the geriatric center because of difficulties walking, and because of rapidly progressive gait and balance disorder, recurrent falls, resting tremor and loss of appetite for three weeks. Her medical history revealed hypertension, chronic obstructive pulmonary disease, urinary incontinence, and she had been taking quinapril 20 mg/day, carvedilol 12,5 mg/day, oxybutynin 10 mg/day for a long time. No other disease or new medication was recorded. She had no symptoms of neuropathy, osteoarthritis or myelopathy, and daily fluctuations, visual hallucination or delusions according to the information obtained from her family. She was admitted to the geriatric service.

On physical examination, her vital signs were stable. Assessment of mental condition showed cognitive impairment, severe time and place disorientation, and disturbances of attention. She presented with asymmetric rigidity (right upper limb), right hand tremor, bradykinesia, and severe postural instability. She did not have any cerebellar or pyramidal signs. Examination of other systems was normal. The cranial MR was also performed and revealed cerebral atrophy and subcortical chronic ischemic alterations with no acute neurological signs. Biochemistry showed normal liver, thyroid, and kidney functions, with no electrolyte imbalance or vitamin deficiencies. Her hemogram parameters were normal, but erythrocyte sedimentation rate was 38 mm/h and C-reactive protein was 14.1 mg/dl. The urinalysis showed no evidence of infection, proteinuria, or

hematuria. Based on these findings regard as of acute and rapid progressive course, she was diagnosed with typically Parkinson's Disease (PD). Levodopa was administered, and her parkinsonian symptoms partially were resolved. However, three days later, she developed fever, anemia, elevation of CRP (207 mg/L), but there was no evidence on infectious disease and level of procalcitonin was low (0,52 ng/ml). The ratio of urine protein to creatinine was shown to be 621.8 mg/dl in the new urinalysis. She developed pancytopenia on the 6th day of hospital admission. Following these findings, she had a test for SLE, and it was found that level of anti-nuclear antibodies was negative, but level of anti-DNA antibodies was positive (224,36 U/ml, normal range < 25), serum levels of C3 and C4 were low 0.72 and 0.07 g/l respectively. Thus, she met five criteria for SLE according to the SLICC criteria (7). She was treated with intravenous methylprednisolone 1 g/day for 3 days then with oral prednisone, and there was a considerable improvement in her symptoms. Since all parkinsonian symptoms as well as abnormal laboratory and clinic parameters were resolved with steroid, it was thought that they might be related to SLE; hence, the dose of levodopa was decreased gradually, and stopped. She was discharged with oral prednisone (32 mg/d) and chloroquine. A month later, all her symptoms were improved.

DISCUSSION

Involvement of the nervous system by SLE includes a wide variety of neurologic and psychiatric manifestations. Movement disorders are uncommon, and parkinsonism is so extremely rare with a prevalence of less than 1% that parkinsonian signs are not included in the 1997 American College of Rheumatology revised criteria for classification (5,8,9). In the literature, a few cases reported for parkinsonism linked to SLE were children or adults (5,6), and most of them occured during the course of a defined SLE (5,6). Furthermore, Dennis et al. reported five senior cases with neuropsychiatric lupus: The two presented with a subacute confusional state, another two with dementia, and the other with depression (4). Therefore, as far as we are concerned, our case report is the first to be associated with parkinsonism as the first manifestation of SLE, and completely cured by steroid therapy in an older adult.

Since the underlying pathophysiologic mechanism of SLE-associated parkinsonism is not clearly clarified, there is evidence that some immune system-mediated pathologies, such as anti-dopaminergic antibodies, may induce dopaminergic cell death (10). A study demonstatred that the presence of autoantibodies such as anti-dsDNA was correlated with clinical manifestations of PD (11). In addition, antibodies can result in immune complex deposition or direct injury to endothelial wall, which may lead to decreased basal ganglia perfusion due to the vasculopathy in thalamostriate arteries (6). Our case's signs of parkinsonism associated with SLE may have occured due to immune-mediated cytotoxic edema, which supports that the improvement is followed by the immunosuppressive therapy. Even if an immunosuppressive drug is usually a

primary therapy for SLE-associated parkinsonism, levodopa supplementation could be helpful, but symptomatic improvement may be partial, as in our case (12).

In conclusion, the diagnosis of late onset lupus may be more difficult than that of early-onset, and may be overlooked because of insidious onset, low prevalence, and the initial negativity of immunologic tests and the lack of SLE criteria. On the other hand, PD is more common in older adults, which can also make the diagnosis of lupus difficult in this population. Even if it is extremely rare, parkinsonism may be due to SLE in an older adult. Therefore, prior to PD diagnosis or while following patients with parkinsonian syndromes, clinicians must be aware of other SLE criteria. More studies are needed to establish how SLE causes parkinsonian signs.

Description of Author Roles: P Soysal, H Akbas, and E Dutoglu saw the case. P Soysal wrote the paper.

Compliance with Ethical Standards

Ethical Statement: The patient's anonymity is protected.

Declaration of Interest : The authors report no conflicts of interest.

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