CASE REPORT

Obsessive-Compulsive Symptoms as a Manifestation of Neuropsychiatric Systemic Lupus Erythematosus

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Neuropsychiatric syndrome is a common and serious clinical manifestation of systemic lupus erythematosus (SLE), occurring in about half of all patients during the course of the disease. We report here a case of obsessive-compulsive symptoms as an unusual manifestation of neuropsychiatric SLE. A 17-year-old girl with SLE was admitted to a medical center with the chief complaint of recent-onset repeated doubt and repetitive checking, which subsided after treatment. She had recurrent and intrusive doubt about losing her belongings and tried to suppress the thoughts by mental acts of praying and repetitive checking. The central nervous system lupus involvement in this patient was confirmed by brain magnetic resonance imaging, which revealed widespread areas of abnormal high signal intensity over the internal capsules and basal ganglia, and focal lesions at the peripheral parenchyma of the frontal and parietal areas. Single photon emission computed tomography also showed decreased perfusion at the left temporal lobe. This supported the hypothesis that basal ganglia abnormalities could be the common pathophysiology of coexisting obsessive-compulsive symptoms and SLE. Moreover, increased awareness of hidden psychopathology, an accepting attitude, and careful probing for obsessive-compulsive symptoms are important while taking care of SLE patients. [*J Formos Med Assoc* 2008;107(1):68–72]

Key Words: neuropsychiatric syndrome, obsessive-compulsive symptom, systemic lupus erythematosus

Systemic lupus erythematosus (SLE) is a systemic immunologic disease that is characterized by hyperactive autoreactive lymphocytes and inflammation induced by immune complexes. Among the varied clinical features of SLE, neuropsychiatric syndrome is a common and potentially serious manifestation, occurring in about half of all patients during the course of the disease.[1] Four neuropsychiatric syndromes of SLE were defined by the American College of Rheumatology in 1999, and they are anxiety disorder, cognitive dysfunction, mood disorder, and psychosis.[1]

However, compared to other psychiatric syndromes, obsessions or compulsions, which are the key symptoms of obsessive-compulsive disorder (OCD), are not as commonly mentioned in previous studies of neuropsychiatric SLE (NPSLE).[2,3] Briefly, the clinical features of OCD symptoms are feelings of subjective compulsion to carry out some action, dwell on an idea, recall an experience, or ruminate on an abstract topic. Dispelling unwanted thoughts or urges may lead to severe tension accompanied by intense anxiety.[4] Reports suggest that the most common symptoms of OCD

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in SLE are washing, grooming, and checking rituals and/or preoccupation with disease, danger, and doubt.[4]

The etiology of OCD is highly heterogeneous and previous neuroimmunologic studies revealed positive links between OCD and autoimmune diseases, especially among children and adolescents.[5–9] Since OCD is typically a psychiatric disorder and may be more prevalent in autoimmune disease,[3,9] healthcare workers should pay more attention to this disabling neuropsychiatric manifestation while taking care of SLE patients. We report here a patient who had typical symptoms of OCD as a manifestation of NPSLE.

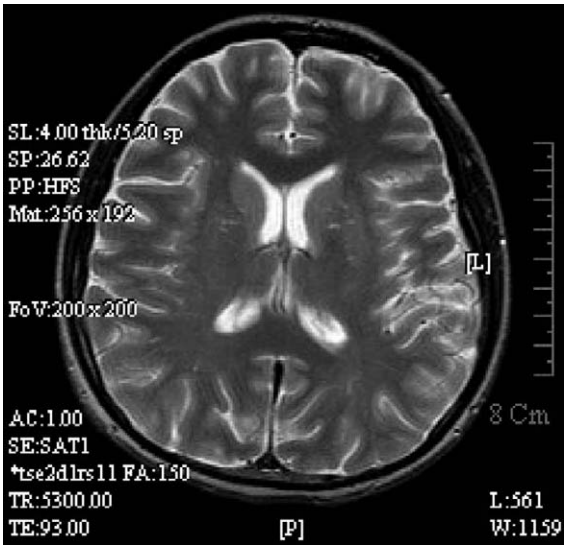

Figure 1. Axial view on T2-weighted brain magnetic resonance (TR, 5300 ms; TE, 93 ms) shows abnormal high signal intensity in the internal capsule and basal ganglia.

Case Report

A 17-year-old girl with SLE was admitted to a medical center with the chief complaint of repeated doubts and repetitive checking. Her birth history, including the process of pregnancy and delivery, had been uneventful. Her early development and history were likewise unremarkable. She was healthy until 4 years ago, when the patient experienced generalized edema that caused weight gain, where her body weight went from 45 kg to 58 kg in 3 months. Symptoms of exercise intolerance and dark papules on the whole body with itching sensation were also observed.

Initial laboratory work-up revealed hematuria, decreased 24-hour creatine clearance, proteinuria, hypoalbuminemia, hypocalcemia, anemia, and low C3 (40.8 mg/dL) and C4 (< 10 mg/dL). A diagnosis of SLE was made according to a positive test for antinuclear antibodies, at a titer of 1:2560, with a homogeneous pattern and associated symptoms, including lupus nephritis and hypertensive encephalopathy-related seizures. Other autoantibodies, like anti-ENA, anticardiolipin antibody, and antiphospholipid antibody, were all negative. Computed tomography (CT) of the head showed multifocal low densities in bilateral high parietal and posterior frontal lobes. Reversible posterior leukoencephalopathy was considered.

For her deteriorating renal function with repeated edema in the next 2 years, the patient received 11 courses of cyclophosphamide treatment and three courses of methylprednisolone pulse therapy. One and a half years ago, she began to receive regular peritoneal dialysis after an episode of pericardial effusion. Chronic renal insufficiency-related anemia was also impressed. Unfortunately, about 1 year before the present admission, she experienced two episodes of generalized tonic seizures with upward gazing and trismus.

After excluding the possibility of infective encephalopathy, central nervous system lupus involvement was confirmed by brain magnetic resonance imaging, which showed widespread areas of abnormal high signal intensity in the internal capsule and basal ganglia (Figure 1) and focal lesions in the peripheral parenchyma of the frontal and parietal areas (Figure 2). Single photon emission CT also showed decreased perfusion in the left temporal lobe. A fourth course of methylprednisolone pulse therapy was then given and epilepsy gradually subsided.

About 1 week before admission, nascent obsessive-compulsive symptoms developed abruptly without identifiable acute stressors. She had recurrent and intrusive doubts about losing her belongings, e.g. her ID card, and became