

# Glucose Hypermetabolism in Contralateral Basal Ganglia Demonstrated by Serial FDG PET/CT Scans in a Patient With SLE Chorea

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**Abstract:** We reported a 24-year-old woman who developed a movement disorder of mouth and limbs during systemic lupus erythematosus. Brain MRI did not find any abnormalities. Increased FDG uptake in basal ganglia was found using FDG PET/CT brain scan. The symptoms remitted after therapy and the activities of the original hypermetabolic regions were down to normal in the follow-up FDG PET/CT scan. This case implies that the pathophysiology of chorea in systemic lupus erythematosus is different from that of chorea in other diseases, such as Huntington disease and chorea-acanthocytosis, in which hypometabolic basal ganglia was found.

**Key Words:** chorea, FDG, systemic lupus erythematosus

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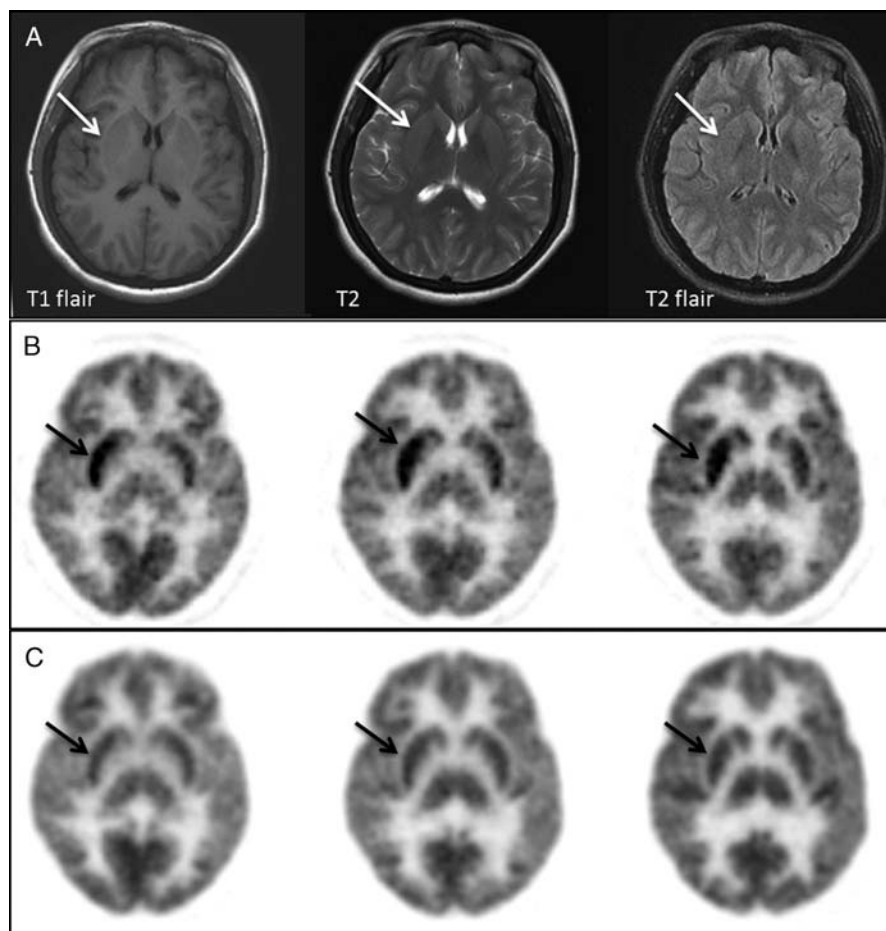
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**FIGURE 1.** A 24-year-old woman, who experienced an episode of SLE-associated pneumonia and hemophagocytic syndrome in the postpartum period at age 23, was successfully treated with intravenous immunoglobulin therapy and blood transfusion. Subsequently, she experienced multiple joint pain, which subsided spontaneously. She presented acute-onset involuntary movements of right limbs and right face 3 months ago. The symptom remitted gradually, but she developed involuntary movement disorder classified as chorea in her left limbs 1 month ago. **A**, Brain MRI showed no significant abnormality in the right basal ganglia (arrows). The first FDG PET/CT brain scan was performed at this time. After fasting for 6 hours, the patient underwent IV injection of 377 MBq  $^{18}\text{F}$ -FDG. Then, FDG PET scan was performed after her sitting calmly with eyes closed in a quiet waiting room for 40 minutes. **B**, Intensely increased FDG uptake in the right basal ganglia (arrows) was shown in transaxial slices of FDG PET. The striata-to-cerebellar cortex and thalami-to-cerebellar cortex ratios of standard uptake value (SUV) were 1.82 (right striatum) and 1.55 (left striatum), and 1.35 (right thalamus) and 1.31 (left thalamus), respectively. Her symptoms remitted completely after intense treatment with a combination of high-dose corticosteroid and immunosuppressive agents, and the FDG uptake in the right basal ganglia returned to normal correspondingly. **C**, Serial transaxial slices of follow-up FDG PET in the same planes showed normal activity in the right basal ganglia (arrows). The striata-to-cerebellar cortex and thalami-to-cerebellar cortex ratios of standard uptake value (SUV) were 1.37 (right striatum) and 1.31 (left striatum), and 1.37 (right thalamus) and 1.34 (left thalamus), respectively. Her laboratory tests kept showing an elevated serum anti-double-stranded DNA and anti- $\beta_2$ -GP1 antibody, elevated LA antibody, and positive serum anticardiolipin IgG during the whole clinical course, which was consistent with the diagnosis of SLE. Choreia is a rare manifestation of systemic lupus erythematosus and in only fewer 4% of patients.<sup>1</sup> Choreia in SLE is recognized as an immune-mediated problem and associated with appearance of aCL antibodies and anti-phospholipid antibody in the serum.<sup>2-4</sup> Small infarctions secondary to vessel occlusion in the striatum were reported,<sup>3,5</sup> although MRI usually showed no significant abnormality in most of these cases with chorea in SLE. Regional cerebral glucose metabolism in 4 patients with SLE chorea was analyzed by Guttman et al. The values for glucose metabolism in the striata did not fall beyond the range established for the control group, but relative striatal glucose metabolism (striatum-to-cortex ratio) in the patients was greater than that in control studies.<sup>6</sup> In our case, the abnormal hypermetabolic striatum associated with chorea was proved by the serial FDG PET/CT scans before and after treatment. FDG PET has been used to study patients with other choreic disorders (Huntington disease [HD], chorea-acanthocytosis, and benign hereditary chorea), and the finding of striata hypometabolism seems to be common.<sup>7-9</sup> This implies that the pathophysiology of chorea in SLE may be different from that of chorea in other diseases.