and her favourable response to limited immunotherapy contrasts with some reports underlying the importance for tumour removal.[1–3] Her favourable evolution appears in line with descriptions of non-paraneoplastic anti–NMDA-encephalitis.[1]

We found an interesting correlate of functional brain imaging, which paralleled her clinical course. To the best of our knowledge, this represents the first sequential description of brain FDG-PET in this context and suggests that her marked limb rigidity might have been mediated by basal ganglia hypermetabolism. Her medication, especially the antiepileptic drugs, with gabaergic action, could alter cortical metabolism but should not increase basal ganglia hypermetabolism. A picture similar to our patient was recently reported in Morvan syndrome,[4] a rare condition due to antibodies to voltage-gated potassium channels characterised by peripheral, central and autonomic nervous system involvement. The only two reports describing FDG-PET in anti–NMDA-R encephalitis showed hypermetabolism in cortical areas, brainstem and cerebellum[3] and a reduced cortical metabolism after clinical improvement[2]; none had basal ganglia abnormalities. Recently, reduction of *N*-acetyl-asparatate in the basal ganglia of a patient in the acute phase of an anti–NMDA-R encephalitis was described,[5] suggesting that modification of basal ganglia circuits may be induced by the autoantibody and lead to extrapyramidal symptoms. In this context, it is possible that the hypermetabolism of the basal ganglia in our patient reflects this aspect; unfortunately, we did not perform magnetic resonance imaging spectroscopy. In our patient, the marked symmetrical basal ganglia hyperactivity with a relative diffuse cortical hypoactivity in the FDG-PET and its progressive normalisation were not correlated with clinical and EEG signs of status epilepticus during the fluoride injections, where the cortex should appear hypermetabolic. We thus suggest that our findings represent the correlation of the extrapyramidal dysfunction and the concomitant cortical hypofunctional state during the active illness.

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CORRECTION

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Refractory central supratentorial hiccup partially relieved with vagus nerve stimulation (*J Neurol Neurosurg Psychiatry* 2010;**81**:821–822). In this paper, the author names were published incorrectly with the first name transposed with the surname. They are correctly listed as follows Pierluigi Longatti, Luca Basaldella, Mario Moro, Pietro Ciccarino, Angelo Franzini.