seen in stroke but with hypometabolism possibly representing a phenomenon of diachisis. Serial FDG-PET examinations showed markedly increased activity in the basal ganglia as compared with that in the cortex when extrapyramidal features were prominent, which normalized after improvement of this movement disorder. In the setting of paraneoplastic neurological syndrome, FDG-PET imaging can be of value from the following standpoints: [1] detection of the occult malignant focus and [2] objective assessment of the presence and extent of the functional abnormality in the brain and correlation of the imaging findings with the clinical features and disease activity. The literature describing the central nervous system findings with PET in paraneoplastic neurological syndromes are primarily restricted to paraneoplastic limbic encephalitis. FDG-PET usually shows hypermetabolism in one or both temporal lobes, and the magnetic resonance imaging (MRI) findings correlate with the FDG-PET scan results in only a fraction of cases. FDG-PET images can provide additional clinical information which is of great value in further patient management.

In conclusion, the FDG-PET more often than the MRI revealed abnormalities in the brainstem, cerebral cortex or cerebellum, some of them clinically silent. FDG-PET might be useful in the evaluation of patients suspected for NMDA receptor encephalitis, but studies to assess the utility for prognosis and treatment planning are necessary.

REFERENCES
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pyridostigmine treatment; and normal chest computed tomography scan. A diagnosis of generalized MG was made and the patient was prescribed pyridostigmine and prednisone. No remission of the disease was observed after one year of treatment; therefore, azathioprine was added to the treatment regimen. The patient also underwent thymectomy and subsequently went into remission. Pathological investigation revealed that the thymus was hyperplastic, but without signs of neoplasia.

At 46 years of age, the patient was still using pyridostigmine and azathioprine, but for one month had been experiencing progressive headaches associated with nausea, vomiting and weakness, with a temperature of 37.9°C. A neurological examination showed mild limitation of ocular movements, bilateral eyelid ptosis, neck stiffness, Kernig’s sign, Brudzinski’s sign and mild paraparesis of both arms and legs (MRC grade 4). A brain computed tomography scan was normal. Lumbar puncture showed an increased opening pressure of 320 mmH₂O, and lumbar cerebrospinal fluid analysis revealed 525 white blood cells/mm³ (6% monocytes, 9% polymorphonuclear cells, and 85% lymphocytes), total protein of 67.4 mg/dl and glucose of 7 mg/dl. Encapsulated yeast suggestive of Cryptococcus sp could be viewed directly (Figure). The latex agglutination method (titer of 1:10,000) was positive for cryptococcal antigen. Cerebrospinal fluid culture growth showed encapsulated yeast compatible with Cryptococcus neoformans. Serum cryptococcal antigen was detectable (titer of 1:10,000). HIV ELISA tests were negative. The CD4+ and CD8+ cell counts were 342 and 192 cells/mm³, respectively, and glucose was elevated (160 mg/dl).

The patient was treated with amphotericin B, which resulted in a marked and prompt improvement of her symptoms and cerebrospinal fluid analysis.

DISCUSSION

The majority of HIV-negative patients who present with CM are apparently healthy, but predisposing factors can be identified in about a third of the cases. Our patient had diabetes mellitus, and was on long-term immunosuppressive treatment, which could have predisposed her towards CM.

Although not surprising in an MG patient under immunosuppression, this is only the third case report of MG complicated with CM. In the two other cases reported thus far, CM followed neoplasms of the thymus (thymic carcinoma), thus suggesting that thymic carcinoma might be a prerequisite for the development of CM in patients with MG. However, although our patient underwent thymectomy, her thymus only showed hyperplasia, and no signs of neoplasm were found. The results from this study support the view that thymic neoplasia may not be the only prerequisite for the development of CM. Thymic dysfunction, caused by neoplasia or the lack of a functional thymus through thymectomy, could itself be a precipitating factor for the development of CM.

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