Since the description of the “shaking palsy” by James Parkinson in 1817, Parkinson’s disease (PD) has been established as the most common extrapyramidal syndrome that has become best understood with distinct correlations with the pathology, biochemistry, and therapy. The core syndrome consists of resting tremor, rigidity, poverty and slowness of voluntary movement, stooped posture, and festination. The modern era of correlating striatal dopamine deficiency with PD was disclosed by Hornykiewicz in 1960 (1), hence the high-dose levodopa therapy was introduced by Cotzias in 1967 with a great success (2). Since then, improvements in pharmacotherapy have been challenged with the use of decarboxylase inhibitors and dopamine receptor agonist. Efforts to uncover the etiology of PD have been intensified in recent years with the discovery of α-synuclein gene (3) and parkin gene (4) in familial cases of PD, but so far the real etiology has not yet been clarified. During this historical course, various disorders which mimic the clinical symptoms of PD have been reported and the term parkinsonism refers to the broad category of these pathophysiological processes. There are many causes for parkinsonism such as postencephalitic, drug-or toxin-induced and other degenerative diseases, so called “parkinsonism-plus”. Among those disorders, parkinsonism in association with CNS infections is a postencephalitic parkinsonism after encephalitis lethargica (von Economo’s disease), which has been well known throughout the world since 1915. But the disease essentially disappeared from the world in 1940 when the diagnosis of encephalitis lethargica was relaxed by the more inclusive term, acute infectious encephalitis. Encephalitis lethargica is a somnolent-ophthalmoplegic encephalitis but the viral agent was never been discovered. But clinical and pathologic features were typical of viral encephalitis with CSF pleocytosis and elevation of protein. The unique symptoms were marked somnolence and ophthalmoplegia-oculogyric crisis. Postencephalitic parkinsonism of von Economo’s type usually appears after the disease, months to years following the acute illness. The pathology in the acute phase was also typical of viral etiology, with cellular infiltration principally in the midbrain, subthalamus and hypothalamus. The autopsy findings of the chronic stage were depigmentation of the substantia nigra and locus ceruleus with nerve cell destruction and glial proliferation in the substantia nigra, oculomotor and adjacent nuclei (5). Parkinsonian syndrome may be seen with a variety of encephalitides, such as western and eastern equine, Coxsackie, measles, chicken pox, Murray Valley and Japanese B encephalitis. In Japan, Japanese B encephalitis-related parkinsonism has been frequently described in detail with clinical signs and autopsy pathology. But these encephalitis-related parkinsonisms are a distinctly different entity from true postencephalitic parkinsonism due to von Economo’s disease. The former syndrome is usually less severe, non-progressive, and is apparent at the time of acute phase without any latent period. Among other infection-related parkinsonisms, neurosyphilis and AIDS have been reported in addition to postencephalitic parkinsonism and transient parkinsonism after acute viral encephalitides and Creutzfeldt-Jakob disease. In this issue of the journal, Murakami et al (6) reported, “Parkinsonian symptoms as an initial manifestation in a Japanese patient with AIDS and toxoplasma infection”.

Reports of extrapyramidal parkinsonian disorders in patients with AIDS due to toxoplasma abscesses or infections have been quite rare (7–10), and particularly so in Japan. Navia et al reported clinical findings in 27 patients with cerebral toxoplasmosis complicating the AIDS. Among those patients, only one case presented with choreoathetosis neurologically. Parkinsonism and hemichorea-athetosis are unusual manifestation of cerebral toxoplasmosis in patients with AIDS. Several mechanisms can be postulated to account for the parkinsonian features in these patients: a) interruption of the nigrostriatal dopaminergic pathways travelling through the internal capsule; b) mechanical pressure or destruction of the striatum or substantia nigra; c) destruction of the striatal postsynaptic neurons; d) transient circulatory disturbances by involvement of the anterior choroidal artery (8). The MRI of their patient revealed a ring-enhanced lesion in the right lenticular nucleus, which could be a responsible lesion to account for his left sided parkinsonism. In their patient, anti-toxoplasmosis therapy was successful for parkinsonian symptoms with the reduction and disappearance of lesions on MRI. Cerebral toxoplasmosis is a curable complication of AIDS. Early diagnosis and prompt therapy are important in preventing mortality and reducing morbidity.

We are now in a new era where we have to consider cerebral toxoplasmosis infection as one of the differential diagnoses in cases with symptomatic parkinsonism when some-what unusual features such as unilateral symptoms are presented.
Genjiro HIROSE, MD, PhD  
The Department of Neurology,  
Kanazawa Medical University,  
Daigaku 1-1, Uchinada-cho,  
Kahoku-gun, Ishikawa 920-0293

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