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İPEK MİDİ
HARİKA G. ÇALIŞKAN
ZAFER TOKTAŞ
AYDIN SAV
DİLEK İ. GÜNAL

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Generalized Choreoathetosis in an Acquired Immune Deficiency Syndrome Patient with Cerebral Toxoplasmosis

Abstract: Many hypokinetic and hyperkinetic movement disorders (e.g. parkinsonism, chorea, myoclonus, dystonia) are associated with acquired immune deficiency syndrome (AIDS) and may sometimes represent the initial manifestation of the illness.

We described a 37-year-old human immunodeficiency virus (HIV)-infected patient with generalized choreoathetoid movements associated with AIDS. Cranial MRI revealed multiple cerebral and cerebellar abscesses. The diagnosis of toxoplasmosis was established by brain biopsy.

Hemichorea and hemiballismus in AIDS patients are pathognomonic findings of cerebral toxoplasmosis. Generalized choreiform movement is a rare condition and related to the bilateral cerebral involvement in toxoplasma disease affecting the subcortical structures.

Key Words: AIDS, choreoathetosis, cerebral toxoplasma abscesses

Introduction

Movement disorders are a potential neurologic complication of acquired immune deficiency syndrome (AIDS) and are identified in 3% of patients with human immunodeficiency virus (HIV). Prospective studies indicate that 50% of patients with AIDS develop extrapyramidal features (1,2).

In the majority of patients, hyperkinetic movements result from lesions caused by opportunistic infections, particularly toxoplasmosis, which damage the basal ganglia connections (2). These types of disorders are generally in hemi-type (1). In the present study, we report a generalized form of movement disorder, which is rare in AIDS patients, due to underlying opportunistic infection.

Case Report

A 37-year-old male patient was referred for consultation because of altered consciousness, inability to talk, and involuntary, irregular and snake-like movements throughout the body with large amplitudes.
Blurred vision and severe headache had developed suddenly about a month before, accompanied by nausea and vomiting.

The neurological examination revealed that the patient was conscious but uncooperative, disoriented, and reacting to voice. Bilateral papilledema was noted on fundus examination. There was no muscular weakness and generalized choreoathetoid movements were remarkable. The deep tendon reflexes were hyperactive with bilateral clonus. The patient did not cooperate with sensory and cerebellar examinations.

The medical history revealed that the patient had a non-vehicular traffic accident 10 years before and had received a blood transfusion abroad.

The cranial magnetic resonance imaging (MRI) showed multiple lesions in both cerebral and cerebellar hemispheres with a suspicion of metastasis (Figures 1, 2). Stereotactic biopsy was performed at our hospital. The biopsy result was consistent with toxoplasmosis (Figures 3, 4).

The routine biochemistry (including liver enzymes, blood urea nitrogen [BUN], creatinine, electrolytes) was within normal limits but erythrocyte sedimentation rate (90 mm/h), anti-HIV and anti-Toxo IgG antibodies were high. The CD4 cell count was less than 50 cells/ml and the patient received antiretroviral treatment and also pyrimethamine for cerebral toxoplasmosis. The antidopaminergic treatment was not effective for controlling generalized choreic movements and the patient died secondary to bacterial respiratory infection.

**Discussion**

Neurological disorders can appear as complications of HIV infection. Movement disorders are increasingly diagnosed as a potential complication of AIDS and may sometimes represent the initial manifestation of HIV infection. While retrospective data from various centers
report an incidence of 2-3% in AIDS patients, when prospective studies were evaluated, the incidence of movement disorders in AIDS patients appears to be higher than previously appreciated (1).

Various hyperkinetic and hypokinetic movement disorders may be seen with HIV infection (e.g. hemichorea-hemiballismus, myoclonus, dystonia, parkinsonism, tremor, dyskinesias). These movement disorders develop either directly due to the HIV infection itself or secondary to the underlying opportunistic infection, but can also be drug-related (drug-induced extrapyramidal syndrome) (1). AIDS patients are particularly susceptible to develop extrapyramidal side effects from some medications such as neuroleptics and anti-emetics. Potential drug interactions between anti-HIV medications such as protease inhibitors may also predispose patients to develop parkinsonism. In the present case, the patient has not been using these types of drugs.

Choreoathetosis in AIDS patients was first defined by Navia et al. in 1986 (3). While some studies define the most common movement disorder in HIV(+) patients as hemichorea-hemiballismus, other reports describe them as the second most frequent after parkinsonism (4,5). Interestingly, hemichorea-hemiballismus is quite rare among movement disorders seen in patients without HIV infection (1). Characteristics of hemichorea and hemiballismus associated with AIDS do not differ from the disorders in an HIV(-) patient (6,7).

The present case had an acute-subacute onset of encephalopathy symptoms with deteriorating consciousness. Generalized choreoathetoid movements appeared about one month after the initial manifestations. Most hemichorea-hemiballismus cases associated with AIDS have lesions in the contralateral subthalamic nucleus or striatum (1,7,8). Generalized chorea was reported more rarely (1,6,8). Multiple lesions affecting thalamus, caudate head, putamen, globus pallidus, midbrain and the internal capsule were related with these generalized involuntary movements.

The hemichorea-hemiballismus in AIDS patients is mostly associated with cerebral toxoplasmosis (found in 7.4% of the cases), and the most common cause is reported as subthalamic toxoplasma abscesses (1,6,9). Some authors state that the presence of hemichorea and hemiballismus movements in AIDS patients is pathognomonic for cerebral toxoplasmosis (3). It is interesting that movement disorders have not been defined in HIV(-) patients with toxoplasmosis (8). Generalized chorea may be seen as a result of bilateral toxoplasma abscesses and may be seen together with HIV-related dementia (7,10).

The intracranial lesions of our patient were found to be consistent with cerebral toxoplasma abscesses (and verified with a biopsy) and the generalized choreoathetoid movements in the HIV(+) patient were deemed to be due to multiple toxoplasma abscesses.

The management of patients with HIV-related hemichorea–hemiballismus includes the treatment of the opportunistic infection, the symptomatic treatment of the movement disorder, and the use of antiretroviral agents. It has been reported that response to therapy was poor and ineffective in controlling the involuntary movements in the generalized chorea seen with HIV encephalitis (10). Our patient was put on antitoxoplasmosis, antidopaminergic and antiretroviral treatment, but the drugs were unsuccessful in controlling the movement disorder. The patient died within the week secondary to bacterial infection.

In conclusion, acute or subacute development of choreoathetoid movements in a young patient without a family history should bring to mind an association with HIV and trigger cranial imaging to determine whether the disorder is due to toxoplasma abscesses.
References


