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A Case of Neuro-AIDS Presenting with Rapidly Progressive Dementia and Parkinsonism with Rapid Response to Treatment

Demans ve Parkinsonizm Tablosu ile Başvuran ve Tedaviye Hızlı Yanıt Veren Bir Nöro-AIDS Olgusu

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Dear Editor,

Cognitive impairment associated with Acquired Immune Deficiency Syndrome (AIDS) by the direct effect of the virus, as well as by opportunistic diseases in the brain, has shown significant changes with the use of combined antiretroviral therapy. Although the brain functions as a "Human Immunodeficiency Virus (HIV)" reserve in spite of treatment, and chronic inflammation and cognitive impairment due to neurotoxic effects of antiretroviral agents can persist in 5-10% of the patients, the incidence of HIV-related dementia, which is the most severe form of infection-related cognitive impairment, is reported to be reduced by about 50% in the post-treatment period (1,2,3,4).

We would like to emphasize the importance of early and accurate diagnosis by presenting a patient with HIV-associated neurocognitive disorder (HAND) who was also diagnosed as having *Toxoplasma* encephalitis, with dementia and parkinsonism and had a rapid response to treatment.

A man aged 54 years presented with symptoms of general malaise, anorexia, weight loss, behavioral change, forgetfulness, and unbalanced and slow walking. He was diagnosed as having primary pulmonary tuberculosis infection one year ago. Symptoms of weakness, fever, and loss of balance did not improve despite the tuberculosis treatment under regular follow-up for six months, and loss of balance worsened within 2 months of the treatment being terminated and symptoms of slowly progressive disorientation, confusion attacks, forgetfulness, anger attacks, and aggression started. He became fully dependent for daily activities during the last three months. Due to stagnation and the subsequent sudden anger attacks,that have recently become evident, he was admitted to the emergency service. The patient had a history of chronic obstructive pulmonary disease, benign prostatic hyperplasia, and 30 packet-years of smoking.

The patient had a cachectic appearance and had herpetic rashes that persisted around his mouth for several months. On neurologic examination, he was apathetic, limited cooperation and impaired orientation to person, place, and time. The fluency of speaking was impaired, and the latency of the response to questions was extended. Bilateral bradykinesia and slow walking in the forefront posture were remarkable. His postural reflexes were impaired, Romberg test was positive, and the tandem walk was incompetent.

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Routine blood and urine tests showed no pathology other than leucopenia (2900 /mm^3), elevated C-reactive protein (143 mg/dL) and erythrocyte sedimentation rate (102 mm/h). Thorax computerized tomography revealed prominent resolution according to the previous findings performed 3 months ago, but piperacillin-tazobactam treatment was initiated for pneumonia due to the observation of new lesions consistent with infection. Contrast-enhanced cranial magnetic resonance (MR) imaging revealed a lesion with contrast enhancement in the left lentiform nucleus that was hyperintense on diffusion-weighted and fluid-attenuated inversion recovery sequences. Signal increases in bilateral white matter, subcortical atrophy, and ventricular dilatation secondary to atrophy were remarkable on T2-weighted sequences (Figure 1, 2). Moderate protein elevation (71 mg/dL) and 50 leukocytes / mm^3 in cell count, predominantly monocytes, were observed in cerebrospinal fluid analysis; no significant pathology was observed in cultures and viral serology.

Anti-HIV antibody positivity was detected when the patient was evaluated for immunodeficiency because he also had a diagnosis of tuberculosis. The HIV confirmation test with Western Blot technique was positive. Simultaneous serum CD4 count was 52 /mm^3 and HIV-RNA level was 886 000 copies/mL. Serum anti-*Toxoplasma* IgG was positive and anti-*Toxoplasma* IgM was negative. The contrast-enhanced lesion on cranial MR imaging was evaluated as *Toxoplasma* infection. The positron emission tomography examination for lymphoproliferative diseases was normal. Combined antiretroviral therapy consisting of lopinavir/ritonavir and tenofovir/emtricitabine, and trimethoprim/sulfamethoxazole for anti-parasitic therapy and clarithromycin for chemoprophylaxis were initiated with the diagnoses of AIDS, HAND, and *Toxoplasma* encephalitis.

At the first month follow-up examination, it was learned that he had experienced episodic visual hallucinations, but had less depression and fewer anger attacks. The level of HIV-RNA was reduced to 7340 copies/mL. He scored 14/30 on the standardized mini-mental test (SMMT), which could not be performed due to poor cooperation during the initial admission. There was a marked improvement in the general condition and cognitive level of the patient at the fourth month follow-up examination. He was fully oriented and cooperated, the dysarthric nature of his speech disappeared and his walk was normal. Serum CD4 level was 225 /mm^3 and HIV-RNA level was 125 copies/mL. On the contrast-

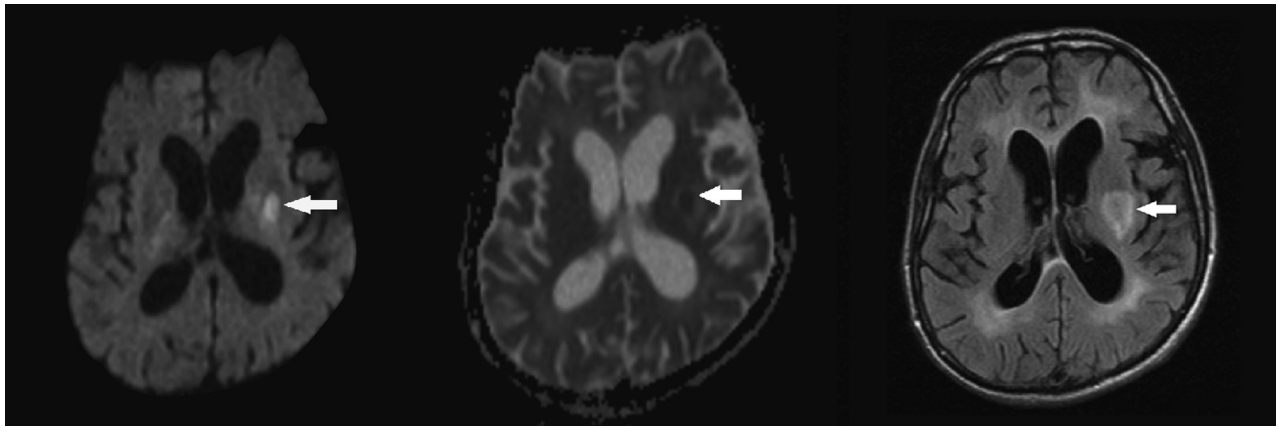

Figure 1. Magnetic resonance imaging performed on admission. Hyperintense lesion in the left lentiform nucleus on axial diffusion-weighted and fluid-attenuated inversion recovery sequences. Subcortical atrophy and ventricular dilatation

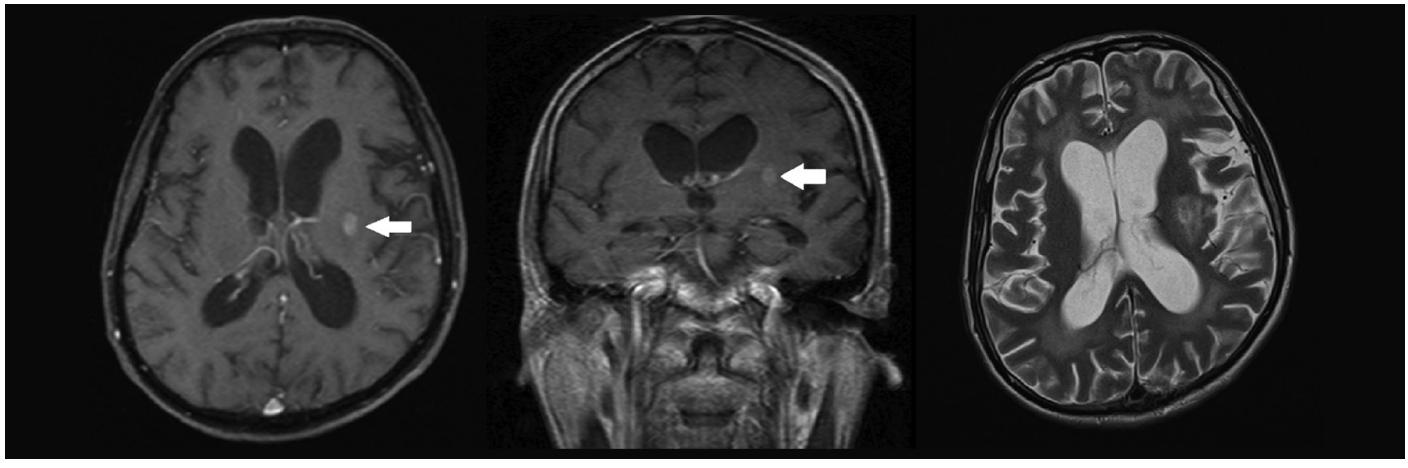

Figure 2. Contrast enhancement of the lesion in the left lentiform nucleus in contrast-enhanced cranial magnetic resonance sections and hyperintensity in bilateral white matter on T2-weighted sequences

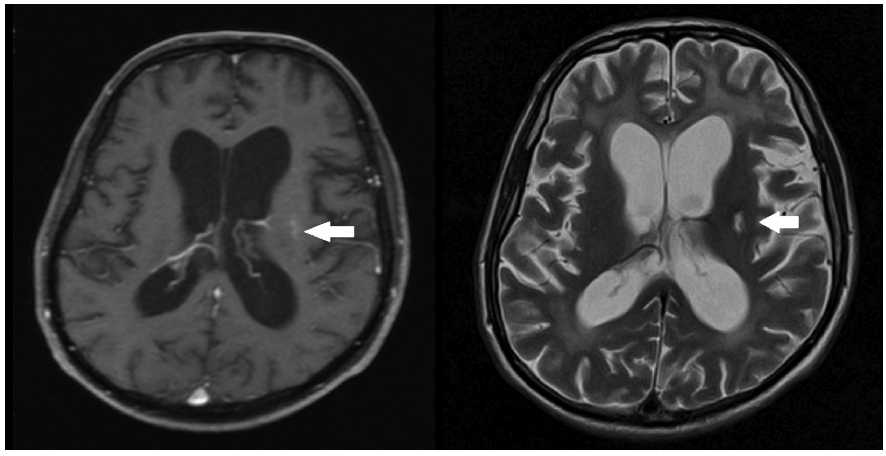

Figure 3. Regression and decreased contrast enhancement in the lesion in the left lentiform nucleus in post-treatment cranial magnetic resonance imaging

enhanced cranial MR examination, the lesion on the left lentiform nucleus was observed as regressed (Figure 3). The SMMT score was 29/30. His relatives stated that his initial symptoms of agitation, irritability, inappropriate affect, and aggression were completely resolved.

It is advisable to confirm the diagnosis of HAND, which has 3 subtypes as asymptomatic neurocognitive disorder, HIV-related mild cognitive impairment, and HIV-related dementia, in case accompanying conditions such as delirium, depression, substance use, central nervous system (CNS) infection, CNS tumor, and cerebrovascular disease are either excluded or severe findings cannot be explained by the accompanying condition (5). In our case, we believe that the findings were so severe that they could not be explained only by the accompanying *Toxoplasma* encephalitis.

The increasing rate of HIV infections in the world has brought the possibility of HAND being among the most common neurocognitive disorders seen in young people. The availability of a specific therapy increases the importance of early and accurate diagnosis. It is of great importance that HIV infection should be considered in patients with neurocognitive findings that give rise to suspicion of immunosuppression, as in our case, or in patients with atypical neurocognitive impairment without history of immunosuppression. In addition, medical history and physical examination should be detailed in terms of neurocognitive evaluation by follow-up departments in patients with HIV infection but without clinical symptoms.

Ethics

Informed Consent: Consent form was filled out by all participants.

Peer-review: Internally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: Y.A., M.S.S., M.B., B.M., U.Ş.E., M.Y., B.S.K., Concept: Y.A., M.S.S., M.B., Design: Y.A., M.S.S., Data Collection or Processing: Y.A., M.S.S., Analysis or Interpretation: Y.A., M.S.S., M.B., Literature Search: Y.A., M.S.S., Writing: Y.A.

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