

**CASE REPORT**

**OLGU SUNUMU**

**STROKE IN A PATIENT WITH HIV INFECTION**

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**ABSTRACT**

Stroke which is a common complication in Human immunodeficiency virus type 1 positive patients is seen between 1% and 5% in clinical series. Vasculopathy and atherogenesis in HIV are the main pathologic mechanisms of stroke. We report a 63 year old man with sudden onset of a right hemiplegia and who was diagnosed as HIV-related stroke.

**Key Words:** Human immunodeficiency virus type 1 (HIV), stroke, atherosclerosis, dyslipidemia, endothelial dysfunction, hypercoagulability.

**HIV ENFEKSİYONLU HASTADA STROKE**

**ÖZET**

HIV (Human Immunodeficiency Virus / İnsan Bağışıklık Yetmezlik Virüsü) tip 1 pozitif hastalarda inme sıklığı klinik serilerde % 1 ile % 5 arasındadır. HIV pozitif inmeli hastalarda altta yatan esas mekanizma vasküler patoloji ve aterosklerozdur. Biz HIV ilişkili inme tanısı alan, ani başlayan sağ hemiplejisi olan 63 yaşında bir erkek olguyu sunmayı amaçladık.

**Anahtar Sözcükler:** HIV tip 1, inme, ateroskleroz, dislipidemi, endotelial disfonksiyon, hiperkoagülabilite.

**INTRODUCTION**

Stroke was reported in Human immunodeficiency virus type 1 (HIV) positive patients since 1980's (1). The direct effect of HIV-1 on stroke pathogenesis is not clear (2). Vasculopathy, cardioembolism, coagulopathy and opportunistic infections play role on stroke in HIV patients (3). Patients with HIV occur stroke between 1% and 5% in clinical series, although a higher rate (4-34%) is seen at autopsy (4-5).

**CASE**

A 63 year old man who was admitted to Medeniyet University Emergency Service for sudden drop in

front of his house's door and not able to stand up. There was no history of seizures, loss of consciousness, fever, headache, nausea or vomiting. He had no history of hypertension, diabetes, smoking and high alcohol consumption as the vascular risk factors. His relatives expressed that, he had stagnation during last month, he was unable to remember his friends and sometimes he dropped the objects suddenly through his hands.

In his neurological examination, the patient was alert however, on mental status examination he was not oriented to person, place and time. Pupils were equal and reactive to light. The patient had right hemiplegia with strength of 4/5 in the right and lower extremities.

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In addition he also had right facial weakness that involved the muscles of the entire right half suggestive of upper motor neuron facial palsy. Rest of the neurological examination was normal. A complete blood count, erythrocyte sedimentation rate, biochemical parameters, including urea, creatinine, blood sugar, electrolytes, lipid profile, liver function tests were all within normal limits. Thrombophilic parameters were unremarkable. Cerebrospinal fluid biochemistry revealed no inflammatory state, with 6 leukocyte, total protein amount 44.70 mg/dl, glucose level 56 mg/dl and also it was negative for VDRL and Mikobacteria PCR. However, serologic studies revealed HIV infection viral load 127.000 copies/ml, furthermore JC virus (John Cunningham virus) PCR was positive, BK virus (Polyomavirus hominis 1) PCR was negative.

Brain imaging studies showed subacute left pons infarction (Figure 1a-b-c) and bilateral white matter hiperintensities (Figure 2) on Diffusion Weighted Image (DWI) and with corresponding hypointensity on apparent diffusion coefficient (ADC) map. Carotid Doppler ultrasounds, transesophageal echocardiography as well as 72 h cardiac and blood pressure monitoring was done. Doppler showed normal carotid arteries. Echocardiogram was unremarkable. Anti-aggregating (acetylsalicylic acid 300 mg) agents was started. The patient was transferred to infectious diseases service where the HIV treatment were continued. After three months of continuous follow up, patient did not show any neurological deterioration but we detected increased white matter hiperintensities on Magnetic Resonance (MR) imaging (Figure 3).

## DISCUSSION

Stroke has received more attention since the human immunodeficiency virus type-1 (HIV-1) became epidemic (6). An African retrospective case control found similar incidence of stroke in the HIV positive patients when compared with HIV negative controls (7). Oppositely, other studies such as Engstrom et al.1989, Park et al.1990 and Qureshi et al. 1997 found a positive relationship between HIV-1 and stroke, especially ischemic stroke (8,9,10). The age of HIV positive patients have stroke younger than HIV negative stroke patients. Ovbiagele et al. reported the median age 42.9 years in 1997 and 48.4 years in 2006 (11).



**Figure 1a.** Diffusion Weighted Image (DWI) shows hyperintensity as subacute left pons infarction.

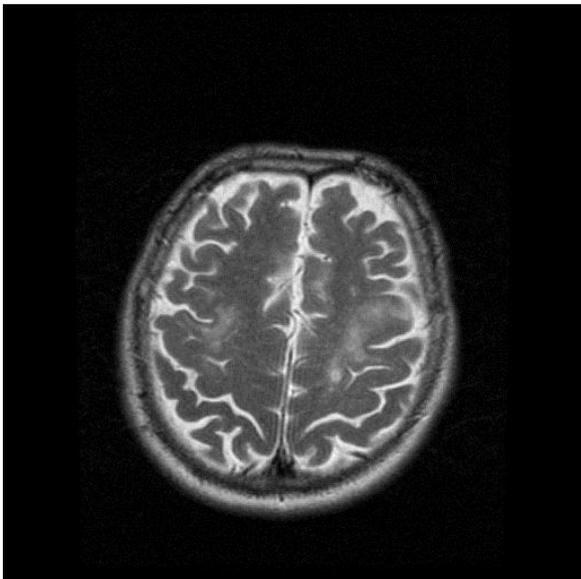


**Figure 1b.** Apparent Diffusion Coefficient (ADC) shows hypointensity as subacute left pons infarction.

When the ischemic stroke classified according to Oxfordshire Community Stroke Project (OCSP) criteria in South African hospital based study, lacunar stroke (n:13), partial anterior circulation stroke (n:33), total anterior circulation stroke (n:11) and posterior circulation stroke (n:7) in positive group(n:64) (12). Comparative results shown that, similar results obtained in the OCSP, except partial anterior circulation stroke

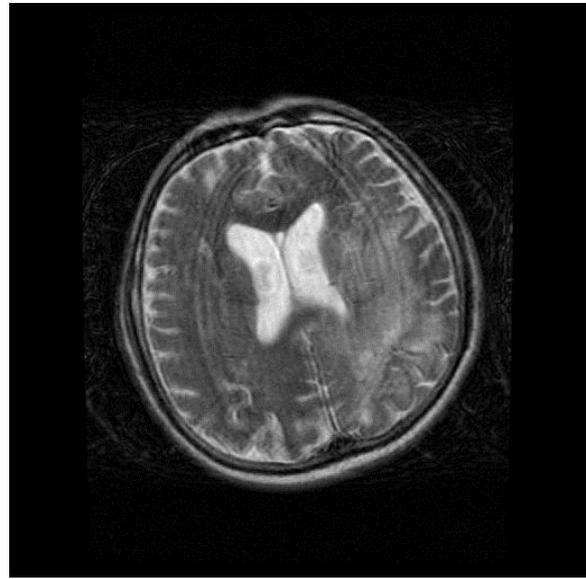


**Figure 1c.** T2 weighted image shows subacute left pons infarction.



**Figure 2.** T2 weighted image shows bilateral white matter hiperintensities.

more often and posterior circulation stroke was less frequent in HIV positive patients (13). However, community based studies are needed for appropriate results rather than small number of patients (3). Results of potential pathologic mechanisms of vasculopathy and atherogenesis in HIV are obtained from HIV transgenic animal models and in vitro human studies (14). HIV-1 may stimulate atherogenesis via increasing the amount of circulating atherogenic immune cells,



**Figure 3.** T2 weighted image shows increased white matter hiperintensities after three months from diagnosis

activating endothelial and immune cells (15). Other mechanisms such as HIV-associated dyslipidemia, endothelial dysfunction, inflammation and hypercoagulability were suggested for relation between HIV infection and cardiovascular risk (16).

Our patient did not have a history of classical risk factors such as hypertension, diabetes, smoking and high alcohol consumption. His laboratory tests showed normal lipid profile. His thrombophilia panel was negative. Also his transeosophageal echocardiogram and carotid doppler ultrasound was normal. Therefore HIV was the only detected risk factor for stroke in our patient.

As a conclusion, it could be suggested that stroke is a more often complication than expected in HIV positive patients. Therefore, in future HIV-1 can be evaluated as a risk factor for stroke etiology. Further study is needed to estimate the convenience and confidence of stroke therapy and prevention in HIV positive patients.

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