Case Report

피질질환의학회지

Imaging Appearance of Human Immunodeficiency Virus Encephalitis on the Diffusion Weighted Images: A Case Report

인간면역결핍바이러스 뇌염의 확산강조영상 소견: 증례 보고

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Imaging finding of human immunodeficiency virus (HIV) encephalitis contain bilateral, symmetric, patchy, or diffuse increased T2WI signal intensities in the basal ganglia, cerebellum, brainstem, and centrum semiovale. In particular, the centrum semiovale is most commonly involved. Most of the HIV encephalitis cases are accompanied by brain atrophy. No previous study has reported symmetric increased signal intensity at the bilateral centrum semiovale without brain atrophy on diffusion weighted images in HIV encephalitis patients. Here, we report a case of this. We suggest that radiologists should consider the possibility of HIV encephalitis if there are symmetric increases in signal intensity at the bilateral centrum semiovale on diffusion weighted images of patients with a history of HIV infection.

Index terms
Human Immunodeficiency Virus
Encephalitis
Diffusion Magnetic Resonance Imaging

INTRODUCTION

Human immunodeficiency virus (HIV) can cause many neurological diseases. HIV-related neurological disorders can be classified as direct and indirect complications. The former are complications caused by the virus itself such as acquired immunodeficiency syndrome (AIDS)-related dementia, HIV-related seizures, and distal symmetric sensory polyneuropathy. The indirect complications include autoimmune disorders, opportunistic infections, and malignancies (1).

HIV encephalitis is a direct complication. HIV, being neurotrophic, causes encephalitis in up to 60% of patients with AIDS (1). HIV encephalitis is a direct consequence of HIV infecting the brain itself. AIDS patients with HIV encephalitis frequently present other AIDS-related intracranial abnormalities, including other infections. However, HIV encephalitis should not combine with other opportunistic infection evidence.

Although the pathophysiology of HIV encephalitis and the direct effects of this disease on the brain remain poorly understood, demyelination and gliosis preferentially affect the deep white matter of the centrum semiovale (2). Most studies of HIV encephalitis cases have reported findings based on the T2 weighted and fluid attenuated inversion recovery (FLAIR) images (1, 3-5). To our knowledge, there are no previous reports of HIV encephalitis appearing on diffusion weighted images. Here, we report on findings of diffusion weighted images from a patient with HIV encephalitis.

CASE REPORT

A 47-year-old man presented at the emergency department with a complaint of weakness in both lower extremities and a previous episode of mild confusion. He did not have any signs of infection and his vital signs were stable. His past medical, social, family history was unremarkable except for being ad-
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mitted to our department of pulmonology for treatment of pneumonia two months ago. He had been diagnosed with atypical pneumonia, probably caused by Candida albicans that was detected by a sputum fungal culture test.

While being treated for pneumonia, the patient's pneumonic symptom did not significantly improve and treatment was prolonged. During pneumonia treatment, the patient was discharged from hospital one month ago. After being discharged, the patient developed mild confusion that was slowly aggravated. After this episode, the patient experienced weakness in both lower extremities and came to our emergency department three hours after this condition developed.

We performed magnetic resonance imaging (MAGNETOM Sonata 1.5 T; Siemens, Berlin, Germany) to identify any brain parenchymal abnormalities. There was diffuse "hazy" increased signal intensity at the bilateral periventricular white matter, centrum semiovale area, and both basal ganglia, mainly in the posterior rim of the internal capsule, on the diffusion weighted images (Fig. 1). There was also diffuse bilateral increased signal intensity in both basal ganglia, mainly in both posterior internal capsules, both frontoparietal centrum semiovale, midbrain, pons, and the middle and superior cerebellar peduncles on the T2 weighted and FLAIR images (Fig. 2). Additionally, there was no parenchymal enhancement or enhancing mass lesion on the enhanced T1 weighted images. The radiologist performing the imaging procedures hypothesized that the primary cause of the patient's symptoms were toxic reagents or a metabolic disorder. However, the patient had no specific history of underlying metabolic diseases or exposure to toxic compounds except for medication for pneumonia.

The attending physician speculated that the patient was suffering from HIV infection; given the patient's history of atypical pneumonia and prolonged treatment. Serologic testing found decreased white blood cell counts and levels of CD4-positive T cells that fell below the normal range. The attending physician conducted serologic testing for HIV antibodies and confirmed that the patient was HIV-positive. The physician subsequently conducted a lumbar puncture to evaluate the cerebrospinal fluid (CSF). The CSF serology test revealed a high titer of HIV RNA (692,000 copies/mL) indicating high HIV virus activity. It was therefore concluded that infection with HIV resulted in encephalitis. There was no evidence of other opportunistic infections related to AIDS, including infection with the JC virus, which causes progressive multifocal leukoencephalopathy (PML) that mimics HIV encephalopathy. Furthermore, there was no evidence of the other opportunistic infective neurologic disorders on the magnetic resonance images.

Based on the CSF study and imaging results, the patient...
was diagnosed with HIV encephalitis. The involved sites were consistent with HIV encephalitis, except for cerebral atrophic changes. The physician recommended anti-retroviral treatment, but the patient rejected his diagnosis, refused treatment, and was discharged from the hospital. Two months after being discharged, the patient returned to the hospital with aggravated neurologic symptoms including weakness in both lower extremities and newly developed voiding difficulties. We speculated that the voiding difficulty was also caused by a neurologic bladder disorder which is a complication of HIV encephalitis. The patient decided to undergo anti-retroviral treatment.

**DISCUSSION**

HIV is a lymphotrophic as well as neurotrophic virus, and central nervous system (CNS) involvement as observed in the present case, is seen in approximately 10% of individuals infected with this virus. Common neurologic symptoms include headache, dementia, confusion, and decreased memory. Symptoms suggestive of a mass lesion may also be present. Various types of organisms that may affect opportunistic CNS infection include *Toxoplasma gondii*, cytomegalovirus, herpes simplex virus, *Cryptococcus neoformans*, *Coccidioides immitis*, *Aspergillus fumigatus*, *Treponema pallidum*, mycobacteria, *Mucor*, and *Candida albicans* (3).

HIV encephalitis is caused by direct HIV infection of the brain itself. There is no evidence of other opportunistic infection of the CNS. Magnetic resonance imaging has provided in vivo evidence of white matter abnormalities in HIV patients (6).

Magnetic resonance imaging results of patients with HIV encephalitis have been published and some common features have been identified. First, visible supratentorial atrophy is commonly observed. Combined infratentorial atrophy is seen in some HIV encephalitis cases; although, this is relatively rare. A serial imaging study of the patients with HIV encephalitis conducted over several months to a few years may reveal progression of cerebral atrophy (4). Except for rapid progression, the features of cerebral atrophy are nonspecific and cannot be differentiated from other atrophy caused by other factors. In a previous autopsy study of HIV encephalitis, HIV was found to predominantly affect the deep white matter and, to a lesser degree, the subcortical white matter while the cerebral cortex was relatively spared (4). Another typical imaging

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**Fig. 2.** Diffusion weighted images. There was increased signal intensity on the diffusion weighted images at the bilateral frontoparietal centrum semiovale (A), and both basal ganglia, mainly in both posterior internal capsules (B); this was consistent with the increased area of T2 signal. Diffusion weighted images suggested active and acute brain parenchymal HIV activity.

Note.—HIV = human immunodeficiency virus
finding of HIV encephalitis is diffuse “hazy” increased signal intensity at the bilateral periventricular white matter, centrum semiovale, and both basal ganglia on T2 weighted and FLAIR images. Diffuse damage to the cerebral white matter is one of the most frequent neuropathologic feature of HIV infection and is particularly prominent during advanced stages of the disease (4, 5).

Our patient did not have any cerebral white matter atrophic changes compared to patients of the same age. A previously noted, most patients with HIV encephalitis have cerebral atrophy with increased signal intensity on diffusion weighted images. To our knowledge, diffusion weighted images for HIV encephalitis patients have not been presented in previous reports. We assumed that there is a reason why the diffusion weighted images of HIV encephalitis patients have not been published. Most cases of HIV encephalitis occur during chronic and advanced stages of HIV infection, so diffusion weighted images are not required or even performed, and in addition no significant fluid diffusion restriction. Therefore, there is no increased signal intensity on diffusion weighted images of HIV encephalitis patients. In our case, the patient had not been previously diagnosed with a HIV infection, high RNA copies in the CSF study, and there was no cerebral atrophy, so we assumed that this case of HIV encephalitis was more acutely advanced. Cerebral atrophic changes require sufficient time (at least several months) to occur, so we believe that our case involves rapid, acutely advanced encephalitis. Acutely advanced encephalitis causes neuronal destruction with fluid diffusion restriction, as well as increased signal intensity on diffusion weighted images.

If there are asymmetric patchy and punctuate white matter lesions on the magnetic resonance images in an HIV patient, several other neurologic disorders should be considered. Asymmetric focal white matter lesions may also occasionally be seen in cases of toxoplasmosis, lymphoma, or PML. When a focal white matter lesion appears on MR imaging, contrast enhanced magnetic resonance imaging may be useful for further evaluation, since toxoplasmosis and lymphoma usually cause enhancement, while HIV encephalitis and PML do not (5).

We should consider the differential diagnosis of PML, which can mimic clinical and imaging features of HIV encephalitis. PML is another disorder that shows evidence of an opportunistic CNS infection by the JC virus in HIV patients. PML is associated with more asymmetric involvement and patchy increased signal on T2 weighted images. However, a CSF study for detecting JC virus (7) is needed to confirm a diagnosis of PML.

In summary, this is the first report describing the bilateral increased signal intensity on diffusion weighted images of an HIV encephalitis patient showing possible acute disease onset. Based on this case, we suggest that radiologists should consider the possibility of HIV encephalitis if there is symmetric increased signal intensity at the bilateral centrum semiovale on diffusion weighted images. This is particularly applicable for patients with a known HIV infection.

REFERENCES

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인간면역결핍바이러스 뇌염의 확산강조영상 소견: 증례 보고

임훈철1 · 유인규1 · 오건세2

Human immunodeficiency virus (이하 HIV) 뇌염의 영상 소견은 양측성, 대칭성으로, 미만성 혹은 반점상으로 양쪽 기저핵, 소뇌, 뇌간, 난형 중심에 T2 신호 강도가 증가된다. 특히 가장 흔하게 난형 중심을 침범한다. 대부분의 HIV 뇌염 증례들은 뇌의 위축을 동반한다. HIV encephalitis 환자에서 뇌 위축 없이, 양측성으로 난형 중심에 신호 강도가 증가된 확산강조영상은 보고된 바가 없다. 따라서 우리는 이 증례를 보고하며 또한 HIV 감염에 대한 임상 정보와 함께 확산강조 영상에서 양측성으로 난형 중심에 신호 강도가 증가한다면 영상의학과 의사의 급성 HIV 뇌염의 가능성을 고려할 것을 제안한다.

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