



A Case Report on HIV-Associated Dementia in a 28-Year-Old Man

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Abstract

HIV-Associated Dementia has since the 1980's been identified as a part of the AIDS complex. While it was once a common finding in AIDS patients, it has been infrequently diagnosed in developed nations since the introduction of highly active anti-retroviral therapy (HAART). We present a case of a 28-year-old man with an 11-year history of poorly controlled HIV who was found to have HIV-Associated Dementia.

Keywords: Therapy; Diabetes mellitus; Anaesthesia

Introduction

A 28-year-old Caucasian man with a history of HIV, Diabetes Mellitus, peripheral neuropathy, recurrent osteomyelitis of both feet, and partial amputation of several toes was admitted to a university hospital for treatment of an apparent infection of his feet. The patient complained of a two-week history of yellow fluid discharge from toes. Upon examination, the patient appeared to be in pain, but was afebrile and normotensive. There was swelling, redness, and broken skin with frank pus discharging from his 2nd and 3rd toes of the right foot and the 3rd and 4th toes of his left foot. Of note, the patient had previous amputations of his 4th and 5th toes of his left foot and his distal 2nd and 3rd toes of his right foot. In addition, the patient demonstrated complete loss of sensation to light touch distal to his mid-calf bilaterally, with inconsistent preservation of deep touch (pressure) sensation from his mid-calf to his ankles bilaterally. The patient also had near complete anaesthesia to deep touch on all surfaces of both of his feet including his toes.

Case Report

The patient was subsequently admitted into the hospital and found to have an HIV viral load of 170,000 and CD4 count of 435. His TSH and B12 are within normal limits. Fluorescent treponemal antibody absorption (FTA-ABS) test is negative. HbA1c level is 15.3. Radiographs of both feet demonstrate erosion of distal metatarsals and amputation of toes with no fractures. Patient was admitted to a medical floor and started on multiple IV antibiotics to treat what was determined to be cellulitis of both feet. His urine toxicology was negative for all substances tested. The next day, a surgical team debrided the patient's infected toes at bedside and determined no further amputation of toes was warranted. He was instructed not to walk until physical therapy could begin working with him. During that night, the patient refused his IV medication, exhibited agitation and told his nurse he would be "better off jumping out this window." The next day (hospital day 3), he was evaluated by a Psychiatry team for possible suicidal ideation.

Over two days of psychiatric evaluation, patient consistently failed to meet criteria for a mood disorder or other psychiatric illness and did not represent a danger to self or others. His agitation resolved following the application of a nicotine patch at the end of day 3. Throughout days 3-5 he consistently demonstrated to both the primary and psychiatry team an increased response latency, slow speech, frequent confusion during questioning, poor insight into his reasons for hospitalization, and inability to remember accurately recall recent events in his life. Cognitive testing was performed by using the Montreal Cognitive Assessment (MoCA), and the patient performed in the range of moderate to advanced dementia. He was discharged to home on day 5 and lost to follow-up.

Discussion

HIV-Associated Dementia has since the 1980's been identified as a part of the AIDS complex [1]. In more recent years, further differentiation into diagnostic grades of neurocognitive disorder have been established, making it easier for a patient to meet criteria for an HIV-associated Neurocognitive Disorder (HAND) when symptomatology meeting the severity of dementia has not been reached. But since the advent and widespread adoption of Highly Active Antiretroviral Reverse Transcriptase therapy (HAART) in the 1990's, patients in developed nations have more infrequently developed dementia as a sequela of untreated or poorly treated HIV infections [1]. HIV-Associated dementia carries an insidious onset and can be the first presenting sign of an HIV infection in 4% to 15% of HIV patients [2]. It does not appear with higher incidence in any gender or ethnic category, and therefore does not appear to demonstrate a genetic predisposition [1]. Its primary risk factor is a high HIV viral load [1]. HAND should be placed lower on the differential in patients who have received early and consistent treatment of HIV [1].

HIV-Associated dementia can be diagnosed via a combination of clinical history, CD4 count, CSF analysis, quantitative HIV blood titers, neuroimaging, neurocognitive testing, and patient interview [1]. It is important to differentiate the three categories of HIV-Associated Neurocognitive Disorders: Asymptomatic Neurocognitive Disorder, Mild Neurocognitive Disorder and HIV-Associated Dementia, the latter of which can only be diagnosed when significant impairment exists in daily functioning regarding self-care or ability to maintain employment [3,4]. The differential diagnosis of HIV-associated dementia includes side effects from anticholinergic medication, B12 deficiency, Cytomegalovirus, CNS Lymphoma, Epstein Barr Virus, Cryptococcus, Delirium, Drug Intoxication, JC Virus, Major Depressive Disorder, metastatic malignancy to brain, Normal Pressure Hydrocephalus, other forms of dementia (vascular, lewy body, alzheimers), Psychosis, Syphilis, Thyroid Dysfunction, and Toxoplasmosis [5].

The pathogenesis of HIV-associated Dementia is thought to be secondary to the direct action of the HIV virus in inducing apoptosis in astrocytes at the blood brain barrier [6]. Once past this threshold, the

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virus has been shown to cause global cortical atrophy [7]. However, studies differ on the location and extent to which HIV also leads to subcortical atrophy throughout the brain. Patients with higher CD4 counts and/or lower plasma viral loads tend to show less loss of brain volume when compared with those who have high viral loads and/or low CD4 counts [5]. The best treatment to slow the advancement of HIV-Associated Neurocognitive Disorders (HAND) consists of appropriate antiretroviral therapy to effectively elevate the CD4 count and lower the HIV viral plasma load [3-5]. It must be noted that even in the setting of a relatively low HIV viral load (<10,000) and normal CD4 count (>500), neurocognitive symptoms may develop and progress over time with an insidious onset and progression [8,9]. The best treated HIV patients, who carry an undetectable viral load, are likely to confer the lowest risk of HAND development and progression [4-10].

Our patient spent approximately 9 of the 11 years since his HIV diagnosis non-adherent to his prescribed antiviral regimen. He had also failed to take his insulin, which led to advance of his diabetic neuropathy of his lower extremities. During this time, he worked primarily as a cowboy herding livestock on horseback and was admitted four times to the University of New Mexico Hospital. All of the admissions involved infections of his feet, including cellulitis and osteomyelitis of his toes, some of which required partial amputation in a past admission. Medical records show that the patient could not recognize that his boots were too small for him due to loss of sensation. It is also worth noting that one admission was preceded by him falling out of his saddle due to what the patient cited as “numb legs;” though he denied striking his head or losing consciousness, a CT Brain was performed, with no trauma found but marked global cortical atrophy noted. Despite the problems he has had as a consequence of his poor glucose control, including a direct effect on his livelihood, he has been unable to recognize the importance of taking his insulin, consistently demonstrating poor insight into the relationship between his diabetes and his foot infections during psychiatric interview despite repeated explanation. Though he had made what had been interpreted as a possibly suicidal statement, repeated interview established that these remarks had been an expression of hyperbolic frustration and that no intent for self-harm existed. He found it difficult to answer questions about his recent life history, often appearing confused. He provided differing answers when the same questions were asked on subsequent days, and these answers also differed from those obtained from his personal friend and sister. It should be noted that the recollection of the friend and sister were similar to each other and consistent with the patient’s medical history of prior hospitalization. UTox has been negative for all substances at every admission. He was also worked up for all of the other medical and psychiatric causes listed above in the differential diagnosis. Though he had a mild cellulitis at time of admission, he was afebrile, alert, oriented, and it was determined by both his primary medical team and consulting psychiatry team that his medical condition did not impact his cognitive functioning.

Furthermore, no change in cognitive functioning was noted between his initial psychiatric evaluation and his final day of admission, by when his cellulitis had been resolved. For these reasons, though not described in the literature in a person of his relatively young age, he was diagnosed with HIV-Associated Dementia.

Conclusion

Because of this patient’s inability to properly understand the need to care for his diabetes or the consequences of not taking his insulin, which have resulted in numerous osteomyelitis infections of his feet and inability to work, he was evaluated for neurocognitive disorder in the context of long-standing and poorly treated HIV infection. He had difficulty understanding questions about his recent life history and demonstrated confabulation in his inconsistent responses. His performance on the MoCA was in the range consistent with dementia, and he could not be made to understand the importance of controlling his diabetes to avoid future foot-related infections and hospitalizations. This, added with the impact on his ability to work, crossed the diagnostic threshold from mild neurocognitive disorder to dementia. There is no better explanation for his dementia than his poorly controlled HIV with high viral load. As a result, we discovered it possible for someone as young as 28 years old to demonstrate HIV-Associated Dementia.

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