Case report: 29-year-old male with Hemichorea-hemiballismus as the initial manifestation caused by cerebral Toxoplasmosis with diagnostic of HIV without treatment

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Abstract:

Background: Hemichorea and hemiballismus are rare movement disorders caused by central nervous system toxoplasmosis.

Objective: Describe the case of a male patient with treatment-naïve HIV who initiated with right hemichorea and hemiballismus.

Case report: we present a 29-year-old male known with history of treatment naïve HIV. He initiated with right hemichorea and hemiballismus to the emergency room. Neurological examination was unremarkable except for the presence of abnormal movements characterized by non-rhythmic, large-amplitude, violent and sometimes choreiform movements in the right side of the body. Brain MRI demonstrated a ring-enhancing lesion in the left basal nuclei. CNS toxoplasmosis was suspected, and further serologic results were positive. Treatment with trimethoprim-sulfamethoxazole and pyrimethamine/clindamycin was initiated, as well as antiretrovirals. Haloperidol, aripiprazole, and clonazepam improved abnormal movements. Further MRI demonstrated radiological improvement and the patient was discharged.

Conclusion: The hemichorea-hemiballismus is a neurological manifestation unfrequently related to CNS toxoplasmosis. Antiretroviral therapy should be started promptly, as well as antimicrobials to improve patient’s outcomes.

Key words: Hemiballismus, Hemichorea, Toxoplasmosis, HIV, basal ganglia, Central Nervous System.
Introduction

Neurologic signs and symptoms may represent the initial presentation of acquired immunodeficiency syndrome (AIDS) in 10-30% of patients. Movement disorders may be the result of direct central nervous system infection by human immunodeficiency virus (HIV) or the result of opportunistic infections. [1] Hemiballismus is characterized by violent and involuntary flinging movements on one side of the body. The prevalence of movement disorders among human immunodeficiency virus (HIV)-infected adult population range between 2% to 44%, with hemichorea-hemiballismus (HCHB) being one of the most common presentations. [2]

Toxoplasma gondii is a ubiquitous, intracellular protozoan parasite that causes central nervous system invasion. The presence of hemichorea–ballism in patients with cerebral toxoplasmosis is not common. However, a subthalamic toxoplasma abscess is the most common cause of hemichorea-hemiballismus in AIDS patients. Although the two phenomena share a similar pathophysiology, proximally predominant, high-amplitude movements are described to ballism, while more distal, lower amplitude movements are often termed chorea. [3]

Berger et al. in 1984 were the first to report involuntary movements in AIDS on the literature. [5] In 1986 Navia et al. presented an extensive work of 27 patients with cerebral toxoplasmosis complicating the acquired immune deficiency syndrome. Regarding abnormal movements, they reported a patient with hemiballismus who also had
choreiform movements of the distal upper and lower extremities and diffuse rigidity and a second had left-sided choreoathetosis. [6]

Case Report

We report a 29-year-old male who presented in our emergency department with a 41-day history of involuntary movements on the right-side extremities. His previous medical history includes smoking of 3 package-year, high risk sexual practices, male-male sex and treatment-naive HIV since four previous years.

His first complaint was acute onset, involuntary and violent movements of his right foot. Three days after, this extended to the proximal right leg. Fifteen days later the symptoms worsened, increasing the amplitude and frequency resulting in difficulty to walk. After a month, the anormal movement progressed to the right superior limb. Also, at this point the patient present dysarthria and facial anormal movements. General examination was unremarkable and neurological examination showed irregular abnormal movements with great amplitude in his right upper and lower limbs (Video1). The movements suggest a pattern of hemiballismus and hemichorea.

Laboratory test reported lymphopenia of 600 cells/μL. MRI demonstrated a ring-enhancing lesion in the left basal ganglia (Figure 1A). Cerebrospinal fluid analysis had increased protein concentration (69 mg/dl) with no cells and further cultures and multiple PCR testing were negative. His HIV1- RNA viral load was 456 920 copies, CD4 cell count 129 cells/μL. His serum toxoplasma IgG was markedly elevated at >150 IU/mL. while syphilis and ant streptolysin O (ASO) titers were negative.
Treatment was started with trimethoprim-sulfamethoxazole (TMP-SMX) 160/800 mg/kg/day and pyrimethamine-clindamycin (P-C) for seven days. After treatment, a new MRI did not result in improvement (Fig.1 B-C). For the abnormal movement the patient had haloperidol 5mg every 8 hours, clonazepam every 8 hours, also amantadine 100mg every 8 hours, considering necessary to add aripiprazole 10 mg once a day. After 6 weeks of treatment, antiretroviral therapy with bictegravir (50mg) tenofovir (200mg) and emtricitabine (25mg) were initiated. The patient had further improvement both clinically and radiologically.

Discussion

Hemiballism (HB) it is a rare disorder that presents unilateral, involuntary, and irregular movements of variable amplitude in the affected host; is considered a more severe expression of hemichorea. [5] Hemichorea (HC) is an involuntary, occasional, rapid, non-patterned series of movements. These two belong to the group of anormal hyperkinetic movements that have the characteristic of a dormant condition when the patient is asleep.

Stroke is the most common cause of acquired hemiballismus. The differential diagnosis includes any focal lesion located generally in basal ganglia structures, but is not exclusive. Hyperglycemic hemiballismus and complications of HIV infection need to be mentioned because they are common and have been recently described in the literature. [7]

Toxoplasma gondii (T. gondii) is an obligate intracellular protozoan. Humans contract the infection via ingestion of the tissue cysts in undercooked meat, oocysts in contaminated...
water or food, and congenitally. [11] Worldwide, an estimated 13,138,600 cases of Toxoplasma gondii infection have occurred in patients with HIV infection; the pooled prevalence among these patients is approximately 35.8%. [9]

Primary T. gondii infection in immunocompetent patients largely remains asymptomatic. In the immunocompromised host, this opportunistic pathogen carries a potential risk of severe disease, especially among people living with HIV (PLHIV) and those with malignancy. [10] Usually is a late complication in patients infected with this virus and usually occurs in patients with less than 200 CD4 T cells/µl [3,4].

The clinical presentation of cerebral toxoplasmosis is nonspecific but usually is subacute neurologic symptoms and signs. The most common presentations include headache, fever, seizures, focal neurologic deficits, cranial nerve palsies, visual disturbances, confusion, and psychomotor or behavioral changes. [9] It is notable that in population with HIV that is known to be highly seropositive to T. gondii, movement disorders comprised only 2.7% of neurological complications. [1] In literature is found there are almost 30 cases report of patient with an anormal movement as first presentation of the infection with Toxoplasmosis.

This case is interesting because Hemiballismus- hemichorea is uncommon. We present a patient with four years of diagnosis of HIV, without treatment. His CD4-positive T cell count was 129 cells/µ. He had clinically right hemichorea and hemiballismus and during sleep these movements diminished.

The preferred initial therapy for cerebral toxoplasmosis according to US guidelines is the combination of Pyrimethamine plus sulfadiazine (P-S). For patients with a history of sulfa
allergy Pyrimethamine plus clindamycin (P-C) is the preferred alternative regimen. The treatment recommendations include TMP-SMX in the first line, when therapies P-C and P-S are not available. [12] The mechanism of Pyrimethamine acts synergistically by inhibiting T gondii proliferation and survival through inhibiting the folate metabolic pathway. The drugs block the synthesis of tetrahydrofolate, which is required by the parasite for DNA synthesis. [13] A final option to consider in severe, medication-resistant hemiballism, is functional neurosurgery. Stereotactic thalamotomy or thalamic deep brain stimulation are other options. [5]

Approximately 30% of T. gondii seropositive AIDS patients with a CD4+ T cell count below 200/µl develop a reactivated, which is lethal if not treated adequately with antiparasitic drugs. Globally, cerebral toxoplasmosis continues to afflict patients for several reasons: late HIV diagnosis, noncompliance with Highly Active Antiretroviral Therapy (HAART) or preventive antimicrobial drugs and virological and immunological HAART failure. [1] In AIDS patients, the reactivation of latent cerebral toxoplasmosis is clinically important.

**Conclusion**

The toxoplasmosis is a common neurological infection but hemichorea-hemiballismus is unusual. Early recognition and prompt treatment of acute symptoms during the initial stage of cerebral toxoplasmosis reduces the risk of neurological sequelae and mortality.
Videos and Figures:

Video 1: Male patient at the entrance of the emergency department, presents movements of hemichorea and hemiballismus on the right side. To watch the video click on the following link:

https://drive.google.com/file/d/1kXIF_DNgKchaAvL8MsQfJcdE2J0ffQPl/view?usp=sharing

Figure 1: (A) MRI at admission, a hyperintensity lesion with contrast enhancing in the left subthalamic nucleus associated with perilesional edema and highlight of the meninges. (B-C) MRI 14 day after treatment without improvement. (D-E) Spectroscopy revealed a peak of choline and n-acetylcysteine

References:


