Reversible movement disorders due to toxoplasmosis as initial manifestation of HIV-AIDS, with sequential MR and video imaging

Antonio Jose Reyes,[1] Kanterpersad Ramcharan,[2,3] Samuel Aboh,[4] Nathaniel Duke[4]

[1]Neurology Unit, Department of Medicine, San Fernando Teaching Hospital, San Fernando, Trinidad and Tobago
[2]Department of Medicine, San Fernando Teaching Hospital, San Fernando, Trinidad and Tobago
[3]Department of Medicine, Surgi-Med Clinic, San Fernando, Trinidad and Tobago
[4]Infectious Disease Unit/Medicine, San Fernando Teaching Hospital, San Fernando, Trinidad and Tobago

Correspondence to
Dr Kanterpersad Ramcharan, kramcharan79@yahoo.com

Accepted 29 May 2016

DESCRIPTION

A previously well 22-year-old African man had cognitive decline for 1 month and involuntary movements for 10 days. Video in segment 1 showed right-sided choreoathetosis. Segment 2, day 6, after 5 days of treatment (table 1) demonstrated less choreoathetosis and brief dystonia of the right foot. Segment 3, on day 10 exhibited normality. The patient scored 5/30 on the Mini Mental State Examination (MMSE). Investigations showed HIV-AIDS associated with secondary syphilis and also a third condition, central nervous system (CNS) toxoplasmosis (table 2). MRI scan of the brain showed cystic and solid areas with hyperintensities and perilesional oedema in some areas on T2-weighted and fluid-attenuated inversion recovery sequences and low signal on T1-weighted imaging in the right parietal-temporal lobes and in the frontal regions, basal ganglia and thalamus bilaterally. The cystic lesions also demonstrated restricted diffusion on diffusion-weighted imaging/apparent diffusion coefficient-weighted images (figure 1A–D). Empirical antibiotic and antiretroviral treatment was, by the use of qualitative Bayesian probability, a necessity since consent for brain biopsy was declined.[1] On highly active antiretroviral therapy, intravenous penicillin for the spirochate syphilis and the antiprotozoal drug, oral trimethoprim–sulfamethoxazole for cerebral toxoplasmosis, the patient gradually improved over the next 4 months, when the MMSE improved to 30/30 and the patient became fully ambulant. Repeat MRI (figure 2A–D), HIV viral load and CD4+ T cell count performed 12 weeks after treatment showed improvement. A significant fourfold rise in serum toxoplasmosis IgG confirmed cerebral toxoplasmosis (table 2).

Damage to the thalamus, subthalamic areas, caudate and/or putamen nucleus and globus pallidus has been postulated as the pathogenic mechanism in movement disorders associated with HIV-AIDS.

To the best of our knowledge, serial imaging demonstrating movement disorders in a patient with HIV-AIDS of new onset, with positive serology for syphilis and confirmed CNS toxoplasmosis, has not been reported previously.[2 3]

Table 1 Medical treatment

	Dosage	Period of treatment
Intravenous drug		
Trimethoprim–sulfamethoxazole	160 mg/800 mg 3 times a day	14 days
Fluconazole	300 mg 2 times a day	5 days
Aqueous crystalline penicillin	4 million units every 4 hours	10 days
Acyclovir	800 mg 3 times a day	14
Haloperidol	5 mg 3 times a day	6 days
Intramuscular drug		
Dexamethasone	16 mg stat followed by 8 mg 3 times a day for 3 days and 4 mg 3 times a day for 3 days	7 days
Oral drug		
Lopinavir–ritonavir	200/50 mg per tablet 2 tablets 2 times a day	4 months
Tenofovir disoproxil-emtricitabine	245/200 mg per tablet 1 tablet daily	4 months
Paracetamol	500 mg 3 times a day	6 days
Pantoprazole	40 mg 2 times a day	6 days
Azithromycin	500 mg daily for 1 week followed by 1 g weekly	4 months
Trimethoprim–sulfamethoxazole	80 mg/400 mg per tablet 2 tablets 2 times a day	4 months
Fluconazole	150 mg daily	4 months
Carbamazepine	200 mg 2 times a day	6 days
Phenytoin	100 mg 3 times a day	4 months

To cite: Reyes AJ, Ramcharan K, Aboh S, *et al*. *BMJ Case Rep* Published online: [*please include* Day Month Year] doi:10.1136/bcr-2016-215676

Table 2 Medical investigations

Blood test	Result	Reference range
White cell count	18.4×10^9/L	$4.5–11.0\times10^9$/L
Venereal disease research laboratory on admission	Reactive	Non-reactive or reactive
Fluorescent *Treponema pallidum* antibody absorption on admission.	Positive	Positive or negative
Elisa for HIV	Reactive	Non-reactive or reactive
Western blot test for HIV	Positive	Positive or negative
HIV viral load on admission	217 839 RNA copies/mL	20–10 000 000 copies/mL
HIV viral load 1-month after admission	48 477 RNA copies/mL	20–10 000 000 copies/mL
CD4+ T cell count on admission	3 cells/μL	410–1590 cells/μL
CD4+ T cell count 4 months after admission	121 cells/μL	410–1590 cells/μL
Antistreptolysin O titer	26 IU/mL	0–200 IU/mL
T. gondii IgG antibodies on admission	3.63	Positive: >1.09
T. gondii IgM antibodies on admission	0.23	Negative: <0.55
T. gondii IgG antibodies 4 months after admission	>250	Positive: >1.09
T. gondii IgM antibodies 4 months after admission	0.0	Negative: <0.55
Herpes virus 1 IgG antibodies	5.00	Index positive: >1.10
Herpes virus 1 IgM antibodies	<0.9	Index negative: <0.9
Herpes virus 2 IgG antibodies	1.30	Index positive: >1.10
Herpes virus 2 IgM antibodies	<0.9	Index negative: <0.9
Other tests		
CSF test (lumbar puncture performed on day 3rd after admission)		
Opening CSF pressure	12 cm of H_2O	6–25 cm of water
Cell count	Nil	0–5 cells/mm^3
Protein	28.3 mg/dL	5–40 mg/dL
Glucose	60 mg/dL	50–80 mg/dL
Culture	No bacterial growth	No bacterial growth or bacterial growth
Cytology	Negative for neoplastic cells	Negative or positive for neoplastic cells
India ink for *Cryptococcus neoformans*	Negative	Positive of negative
VDRL	No reactive	Non-reactive or reactive
FTA-ABS	Negative	Positive or negative
PCR for *herpes virus*, *Cytomegalovirus*, Epstein-Barr virus, *Enterovirus*, *Parvovirus*, *Lymphocytic choriomeningitis virus* and *T. gondii*	Tests not obtained	Negative or positive
Scalp EEG	Normal	Normal or abnormal
Brain/leptomeningeal biopsy	Consent was declined	

CSF, cerebrospinal fluid; FTA-ABS, Fluorescent *Treponema pallidum* antibody absorption; T. gondii, Toxoplasma gondii; VDRL, Venereal Disease Research Laboratory test.

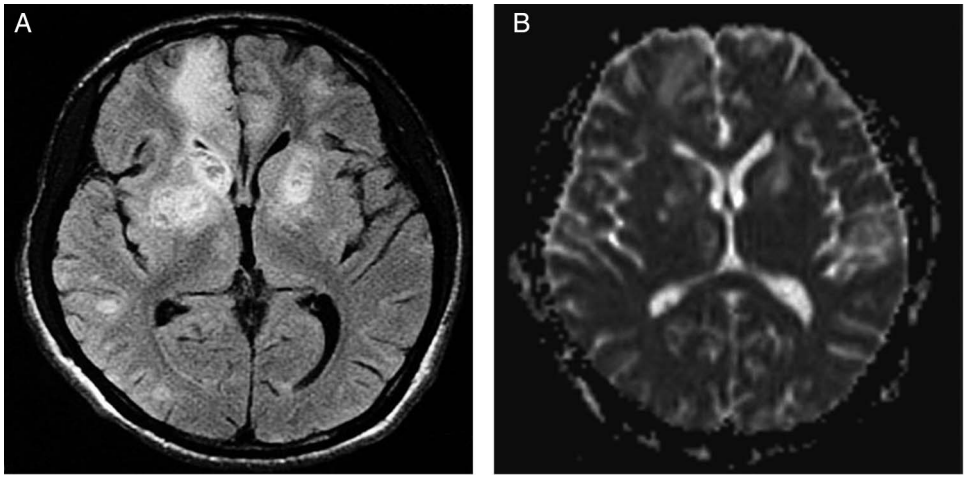
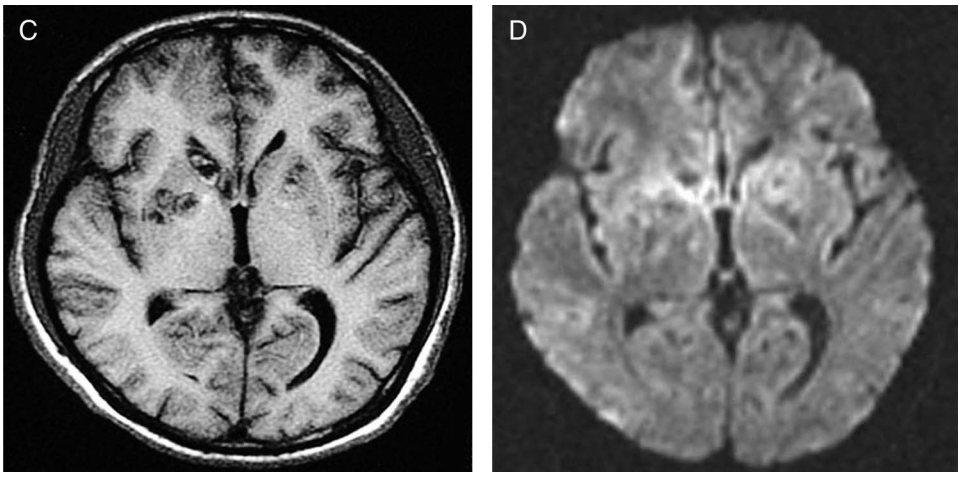


Figure 1 (A) Axial T2-weighted fluid-attenuated inversion recovery MRI view with multiple abnormal hyperintense signals in basal ganglia and thalamus bilaterally, frontal regions and right parietal and temporal lobes suggestive of central nervous system (CNS) toxoplasmosis, but HIV encephalopathy and/or CNS lymphoma could not be ruled out. Some lesions showed mild perilesional oedema. (B) Axial diffusion-weighted imaging with restricted diffusion of the cystic lesions involving both cerebral hemispheres. (C) Axial T1-weighted MRI view with low signals located in basal ganglia and thalamus bilaterally, and in frontal regions and right parietal-temporal lobes. (D) Axial MRI apparent diffusion coefficient (ADC) map with reduced ADC values, which confirms restricted diffusion in the cystic lesions.

Figure 1 Contined



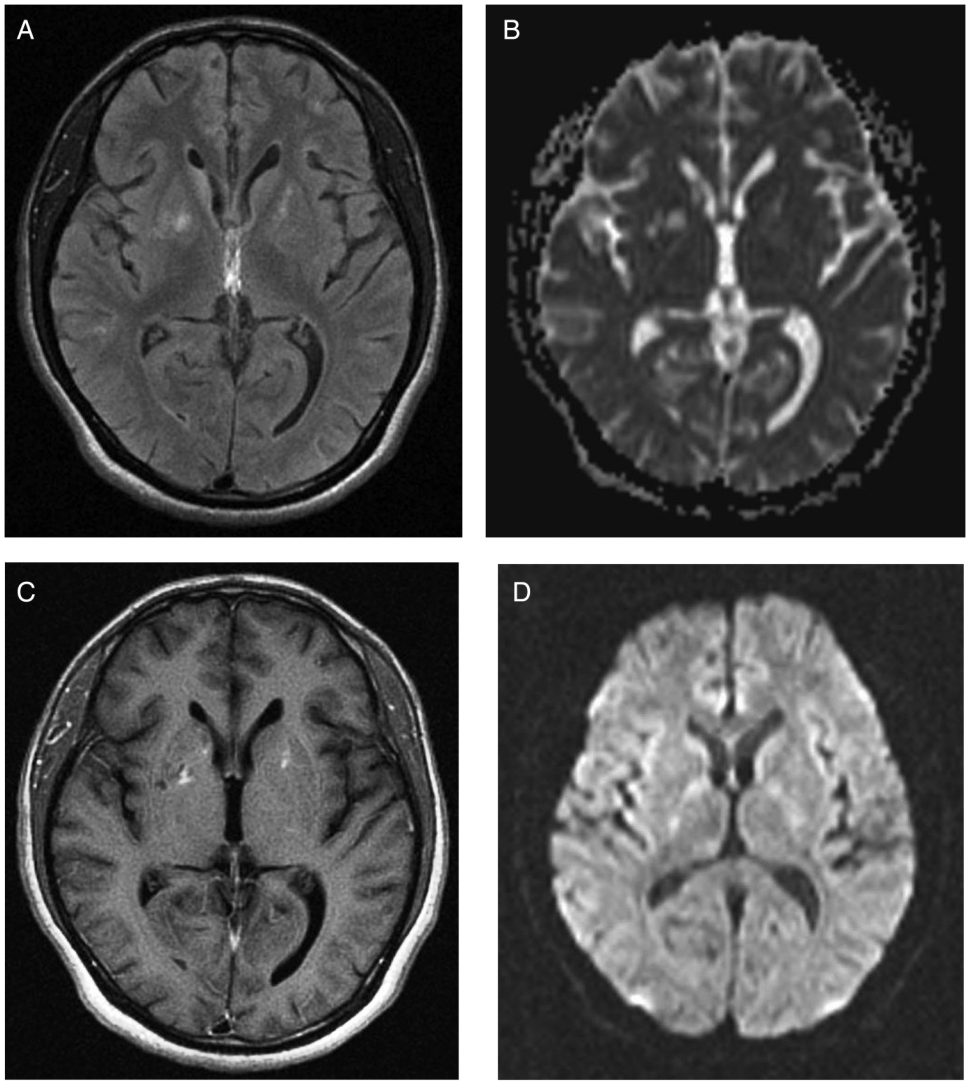
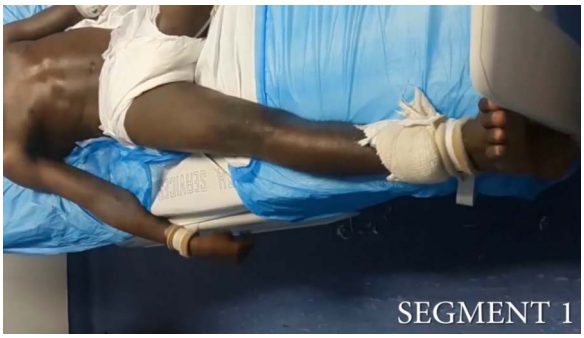


Figure 2 (A) Axial T2-weighted fluid-attenuated inversion recovery MRI view with significant improvement of abnormal hyperintense signals and oedema throughout. Small abnormal hyperintense signals remain in basal ganglia bilaterally, and in frontal regions and right parietal-temporal lobes. (B) Axial diffusion-weighted imaging with restricted diffusion of the cystic lesions involving both cerebral hemispheres with significant improvement. (C) Axial T1-weighted MRI view with low signals located in basal ganglia and thalamus bilaterally, and in frontal regions, and right parietal and temporal lobes, with significant improvement. (D) Axial MRI apparent diffusion coefficient (ADC) map with reduced ADC values, which confirms restricted diffusion in the significantly decreased number of cystic lesions.



Video 1 Segment 1 showed right-sided choreoathetosis;Segment 2, day 6, after 5 days of treatment (table 1) demonstrated less choreoathetosis and brief dystonia of the right foot; Segment 3, on day 10 exhibited normality.

Learning points

- HIV-AIDS related multiple neuropathology is a documented cause of abnormal movement disorders.
- Damage to the thalamus, subthalamic areas, caudate and/or putamen nucleus, and globus pallidus has been postulated as the pathogenic mechanism in movement disorders associated with HIV-AIDS.
- Constrained by the demand of patients for non-invasiveness, or due to unavailability of tests, aggressive empirical antibiotic and antiretroviral therapy is, by the use of Bayesian probability, a practical necessity that can prevent death and disability among patients with HIV-AIDS-related multiple neuropathology.

Acknowledgements The authors would like to thank Dr Fidel Rampersad for assistance with the radiological aspects, Dr Shane Karim for preparation of the video and Ms Sharon Sealy for preparation of the images.

Contributors AJR conceived the idea of this case report. All the authors contributed equally to preparation of the manuscript and approved the final contents.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1 Medow MA, Lucey CR. A qualitative approach to Bayes' theorem. *Evid Based Med* 2011;16:163–7.
2 Tse W, Cersosimo MG, Gracies JM, *et al*. Movement disorders and AIDS: a review. *Parkinsonism Relat Disord* 2004;10:323–34.
3 Sanchez-Ramos JR, Factor SA, Weiner WJ, *et al*. Hemichorea-hemiballismus associated with acquired immune deficiency syndrome and cerebral toxoplasmosis. *Mov Disord* 1989;4:266–73.