HIV-associated neurosyphilis: Report of a fatal case due to fear of work-place stigma

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Abstract
Syphilis and HIV infection are two STI diseases that have bidirectional influence on the clinical course of each other. There is a high risk of neurological extension if syphilis is not diagnosed early or if the patient has a co-infection with HIV. Both diseases have stigma associated with them and could affect the compliance to treatment, as was the case with this young employee of the medical department of a Nigerian tertiary hospital. He was diagnosed with HIV/neurosyphilis co-infection and responded to penicillin therapy, but the fear of stigma at his workplace has made him to abandon hospital treatment for unorthodox therapy, which unfortunately cost him his life.

Keywords: Nigeria, stigma, syphilis/HIV co-infection, workplace

Introduction
Syphilis has a spectrum of presentations — from painless genital ulcer(s) through a non-itchy rash to detrimental effects on, and damage of, the internal organs such as the brain, nerves, eyes and the heart. Syphilis is a re-emerging disease with the current pandemic of human immunodeficiency virus (HIV) infection; it is gaining renewed attention across the world in the form of HIV/syphilis co-infection. The two are acquired through a common risk behavior, and both have a reciprocal impact on the clinical course of each other. Syphilis increases the susceptibility of the host to HIV infection by enhancing HIV transmission through the syphilitic ulcers. It raises HIV viral load and decreases CD4+ cell count, while HIV increases the likelihood of neurological manifestation, which may be asymptomatic or present as acute meningitis, neuroretinitis, deafness or stroke, and higher incidence of a syphilitic relapse after treatment. These bidirectional influences on each other have serious implication on the survival of the patients unless prompt diagnosis is made and effective treatment instituted. We present here a case of a young Nigerian diagnosed to have HIV/syphilis...
A 38-year-old Nigerian general practitioner in the northeastern part of the country was admitted with a 5-month history of low-grade persistent fever, headache and dizziness. He had insomnia and on and off neck stiffness. He had intermittent cough, recurrent nonpuritic skin rashes with progressive weight loss. Cough was sometimes productive of yellowish sputum without pleurisy or hemoptysis. He started self-medication with a series of antibiotics, including anti-TB drugs for the cough, and used topical steroid, sulphasalicylic acid and clotrimazole containing preparations for the rash. When there was no improvement in his clinical conditions, he had his sputum tested for tubercle bacilli and screened himself for antibodies to HIV using proxy names. He tested positive for HIV I and II antibodies but had no bacilli in his sputum. He then self-prescribed and started taking antiretroviral drugs without adopting a particular regimen. Two weeks before his hospital admission, he complained of numbness and heaviness of the lower limbs. He however resisted attempts to get him into the hospital; by the time he consented, he was unable to use his right upper and lower limbs and had become agitated, restless and was talking excessively, though with a slurred speech. He was married to 2 women. He neither consumed alcohol nor smoked cigarettes. Before the current employment, he had worked in the medical unit of the Nigerian custom services, where he had been exposed to unprotected sex for some years.

On examination he was a young adult, wasted and febrile (temperature, 38°C). He had a widely spread healed hyperpigmented scars on his trunk, limbs, the perineum and the genitalia. He had oral thrush, desquamating palms and feet and scars of herpes zoster on multiple dermatomes of his lower back. He was pale and had cervical and axillary lymphadenopathy. He was anicteric and had no pedal edema. He had facial nerve palsy on the right side. He was a pale and had diminished muscle power was 4/5. Both the kerning’s and B rudzinski’s signs were positive and reproducible in spite of his altered level of consciousness. His pupils were equal in size with good light reflex, and the fundi were normal. His chest was clear and respiratory rate was 32 cycles/minute. Pulse rate was 104 beats per minute, blood pressure was 120/70 mm Hg and heart sounds were I and II only. There were no palpably enlarged abdominal organs.

A diagnosis of meningovascular syphilis was made in a background of advanced HIV/ AIDS. He was investigated and the following results were obtained: full blood count— PCV- 27%, WBC- 8.8 × 10^9/L, neutrophils- 57%, lymphocytes- 19%, monocytes-9%, others-11% (normal values: WBC- 4-11 × 10^9/L, neutrophils- 45%-74%, lymphocytes- 16%-45%, monocytes- 4%-10%); erythrocyte sedimentation rate- 84 mm/h (normal, male- Westergren value, 0-20 mm/h). Electrolytes and urea showed sodium- 130 mmol/L, potassium- 3.3 mmol/L, urea- 4.9 mmol/L and creatinine- 103 µmol/L (normal values: sodium-135-145 mmol/L, potassium- 2.9-5 mmol/L, urea-2.5-6.5 mmol/L and creatinine- 53-106 µmol/L). Liver function tests showed both aspartate and alanine aminotransferases to be 5 and 13 IU/L, respectively, and alkaline phosphatase- 94 IU/L (normal values: AST-;0-20 IU/L, ALT- 0-15 IU/L, alkaline phosphatase- 21-93 IU/L), and both total and conjugated bilirubins were essentially normal. Venereal disease research laboratory (VDRL) test was strongly reactive on his serum specimens on two different occasions; so was the cerebrospinal fluid sample. Cerebrospinal fluid (CSF) cell count showed significant pleocytosis comprising lymphocytes- 66 cells/mm³ and monocytes-21 cells/ mm³, and the protein concentration was 72 mg/dL. However, CSF Gram stain was not suggestive of bacterial infection, and culture was negative for any pathogen. Ziehl-Neelson staining of the CSF for tubercle bacilli and fungal culture on sabarud dextrose medium were both negative. Culture of his sputum grew Klebsiella spp, and the chest x-ray showed bilateral soft infiltrates in both lung bases. Serology for toxoplasma gondii was not available at our center. After pre-test counseling, he tested positive for HIV I and II antibodies on two occasions using different enzyme-linked immunosorbent assay (ELISA) kits, and his CD4 cell count was 62 cells/µL. These results were available within 24 hours, safe for the cranial CT that had to be done at another facility hundreds of kilometers away. It was delayed for 10 days because of cost, and it showed nonspecific enhancement of the meninges.

He was started on intravenous (IV) crystalline penicillin 4 mu 6 hourly for 21 days and oral nystatin drops 10,000 units three times daily for 1 week. He received IV 10% mannitol 500 mL over 30 minutes thrice daily for 72 hours; and IV dextrose saline, 1 L 8 hourly. He also had twice daily dosing of combination antiretroviral drugs comprising nevirapine 200 mg, stavudine 40 mg and lamivudine 150 mg. He responded well to these treatments,
his temperature normalized to 36.8°C and cough subsided by the seventh day of treatment. His sensorium became clearer, 7th nerve palsy improved and he was able to communicate meaningfully. He also regained some of the motor function of the right limbs as he was now able to drag the paretic limbs; and by the 16th day of admission, he was able to walk with considerable support. On completion of the antibiotic therapy, he started complaining about the disposition of some of the members of the managing team towards him; and on the 24th day of admission, he concluded that every hospital worker had become known about his diagnosis as evident from the number of workers visiting him on the hospital bed. He was reassured that this was not so but he remained adamant. He was persuaded to have a repeat lumbar puncture on the 29th day of admission; however, his CSF remained reactive to the VDRL serologic test. There were also no remarkable changes in the CSF pleocytosis: lymphocyte count was 62 cells/mm³, monocyte count was 16 cells/mm³ and CSF protein was largely unchanged: 70 mg/dL (normal value-, 15-45 mg/dL). However, ESR reduced to 31 mm/h. After 4 weeks of admission, he requested for discharge and got himself discharged from the hospital against appeals and reassurance from the managing team. He later sought unorthodox care from prayer houses and abandoned the ART. He died a few weeks later.

Discussion

Presumptive diagnosis of neurosyphilis was made in this patient by the positive VDRL serologic test that detected antibody to the non-treponemal antigens in his serum and CSF samples. The treponemal hemagglutination (TPHA) confirmatory test could not be done because the test was not available at our center. We are aware of the high rate of false positivity of VDRL test in HIV/ syphilis co-infection because of the polyclonal B-lymphocytes activation. However, because of the patient’s positive HIV status, his neurological symptoms and signs, along with the positive CSF VDRL test and the CSF pleocytosis suggesting meningeal inflammation, the diagnosis of neurosyphilis (meningovascular type) was upheld in him, in accordance with the WHO guidelines of diagnosing and treating syphilis in resource-poor countries. He was treated, as such, with high-dose intravenous penicillin after excluding other opportunistic diseases (using the available resources) that could present with similar neurological features in HIV-seropositive patients. Tuberculosis was excluded by the negative sputum and CSF smears for tubercle bacilli and the non-suggestive chest x-ray features. Candidiasis and histoplasmosis were also excluded by the negative fungal culture of the CSF, and the brain CT scan partly helped to rule out CNS lymphoma, brain abscess and toxoplasmosis, which could have manifested as space-occupying lesions. The scan also excluded HIV-related leukoencephalopathy. Furthermore, the prompt resolution of the clinical and neurological features following the penicillin therapy helped to narrow the diagnosis to an infectious process, which in this case was neurosyphilis when all the laboratory data were considered.

The symptom complex of fever, headache and neck stiffness together with the unilateral hemiparesis and 7th cranial nerve palsy in this patient pointed to diffuse inflammation of the pia–arachnoid mater along with focal or diffuse involvement of small or medium cerebral arteries. However, the patient’s brain scan only highlighted the leptomeninges without evidence of vascular lesions. This perhaps suggested that cerebral arteritis, the suspected cause of the neurological deficit in this case, had resolved upon antibiotic therapy before the CT scan was done. However, it is important to appreciate the limitation of CT scan of the brain in identifying small cerebral lesions (infarctions) arising from small-vessel arteritis in certain locations of the brain, such as the posterior fossa and the cortical surface of the brain. MRI is better for arterial lesions at these sites; however, the patient did not have this test.

He responded to the antibiotic treatment, as evident by resolution of fever, clearance of his consciousness and improvement in weakness in his right-sided limbs. However, this was poorly corroborated on the repeated CSF analysis by only a marginal reduction in CSF pleocytosis, which is the most sensitive index of response to treatment in neurosyphilis. The short time interval between treatment completion and this test could be responsible for this observation. However, the possibility of persistence of activity of neurosyphilis after standard therapy should not be lost, especially in HIV–co-infected patients.

Fear of workplace stigma and possible discrimination against him should his HIV status be known to his colleagues at work made him to get discharge against medical advice. His perception of this fear was partly genuine because some of the doctors and nurses as well as the laboratory scientists that participated in his care were privy to his diagnosis. This could explain his fear that the confidentiality of his status might have been broken. The uncertainty of what the conduct of his workplace colleagues might be towards him probably made him to deny himself the earlier hospital consultation and indulge in self-medications. In an effort to limit contacts with his colleagues, therefore, he had to withdraw himself.
from the hospital and seek comfort in his priest or imam. This is a typical way most people with stigmatizing ailment would behave, especially if it is a do-or-die situation during chronic illness.[8]

Stigma and discrimination against people living with HIV/ AIDS (PLWHA) are commonplace in Nigeria. Both Christians and Muslims see immoral behavior as the cause of the HIV/ AIDS epidemic.[9,10] This affects attitudes towards them. Some are denied employment or lose their jobs. To stem this tide, there should be wider access to VCT, and infected people should be encouraged to be open about their status. Seeing people speaking out about their HIV status can encourage others to embrace VCT. If people are tested and if they speak out before they are seriously ill, the image of HIV/ AIDS would be changed for the better and many would live positively with it. To achieve this, those who speak out must be protected against verbal abuses. The federal government of Nigeria is vigorously working along this line with the enactment of the HIV/ AIDS workplace policy guidelines in year 2005 to help protect the infected workers from workplace stigma and discrimination.

References


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