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Correspondence

Tuberculous meningitis in a patient with acute human immunodeficiency virus infection

The presentation of acute human immunodeficiency virus (HIV) infection is highly variable,[1] and may include aseptic meningitis, meningoencephalitis and encephalitis,[2–5] which may mask signs of co-existing central nervous system infections such as tuberculous meningitis.

A 44-year-old woman was admitted to our department for fever, confusion and difficulty walking. Fifteen days before, she had been admitted to another hospital with fever, rash and confusion, and rapidly improved. Brain computed tomography (CT) scan was normal and cerebrospinal fluid (CSF) revealed 100 white blood cells (WBC)/mm^3 (90% lymphocytes), glucose 50 mg/dl (blood glucose was normal) and proteins 103 mg/dl. Microscopy and culture were negative. She received empiric antiviral and antibiotic therapy and was discharged 10 days later with a diagnosis of viral meningitis (IgM antibodies [Ab] for Coxsackie virus were positive). As her condition worsened, she was re-admitted to our service, where brain CT scan showed diffuse left thalamic hypodensity and left cortical sulci oedema; CSF revealed increased protein value (179 mg/dl), 179 WBC/mm^3 (85% lymphocytes), and negative microscopy. CSF glucose value was 58 mg/dl (blood glucose was normal). Polymerase chain reaction for herpes simplex virus and *M. tuberculosis* in CSF were negative.

After obtaining the patient's consent, tests for HIV were performed: Western blot was indeterminate, with a positive band for gp 120/160 only, but plasma HIV-RNA level was 40 000 cp/ml and CD4 cell 426/mm^3. Her husband had used intravenous drugs 20 years earlier and had repeatedly refused to undergo HIV testing; later, however, he tested positive.

A subsequent lumbar puncture showed HIV-RNA 2400 cp/ml and decreased glucose levels (36 mg/dl). Empirical anti-tuberculosis treatment with isoniazid, rifampicin, ethambutol and pyrazinamide was started. Repeated CD4 count showed a sharp decrease (178/mm^3) and HIV-RNA a marked increase (>500 000 cp/ml). HIV genotype showed the presence of polymorphisms in the protease region (L63P, V77I). Antiretroviral treatment (ART) with tenofovir, lamivudine and efavirenz was started. The patient's neuropsychological condition progressively improved; brain CT showed a resolution of the earlier lesions and subsequent lumbar puncture showed normal protein and glucose values. CSF culture grew *M. tuberculosis*. Two months later, HIV Western blot was positive for all bands, HIV-RNA was undetectable and CD4 count had increased to 396 cells/mm^3. Anti-tuberculosis treatment was continued for 12 months, always associated with ART.

Our case establishes the need for HIV testing in patients with acute meningoencephalitis and meningitis, even in the absence of a history of risk factors. The diagnosis of HIV infection is crucial—for public health reasons because counselling reduces the risk of transmission, and for individual reasons because early ART improves prognosis by slowing the rate of disease progression. Tuberculous meningoencephalitis can complicate acute HIV infection and can be confused with the clinical presentation of HIV meningoencephalitis. Co-existence is further complicated by the difficulty in diagnosing tuberculous meningitis, and empirical treatment may be warranted.

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Amikacin-induced hypomagnesaemic tetany complicating multidrug-resistant tuberculosis treatment

Amikacin (AM), an aminoglycoside antibiotic, is one of the most efficient second-line injectable drugs available for treatment of multidrug-resistant tuberculosis (MDR-TB). A treatment duration of 6 months is