Neurosyphilis Showing Transient Global Amnesia-like Attacks and Magnetic Resonance Imaging Abnormalities Mainly in the Limbic System

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Abstract

We report a case of neurosyphilis with transient global amnesia (TGA)-like attacks on the first presentation. MRI abnormalities in bilateral limbic systems, including a few lesions in the basal ganglia and thalamus, were identified. Depression and dementia became apparent, accompanied by a high treponemal antibody titer and mild cortical atrophy. Antisyphilitic therapy brought about mild improvement, and the MRI abnormalities decreased. (Internal Medicine 40: 439-442, 2001)

Key words: magnetic resonance imaging, meningovascular neurosyphilis, general paresis

Introduction

In recent years, neurosyphilis has become more prevalent with the increase of immunocompromised hosts (1, 2). Magnetic resonance imaging (MRI) or single-photon emission computed tomography (SPECT) of meningovascular neurosyphilis and general paresis have revealed multifocal lesions due to vasculitis, cerebral cortical atrophy, white matter changes, and the reduction of cerebral blood flow (3, 4). We report a case of neurosyphilis in a non-immunocompromised patient showing transient global amnesia (TGA)-like attacks, and MRI abnormalities mainly in the bilateral medial temporal lobes.

Case Report

A 41-year-old unmarried male truck driver had had venereal exposure at the age of 25 years old. In September 1998, he lost his way home, and was assisted by the police. He did not remember this event. On January 10, the man visited a neurosurgery clinic, and on February 10 he was admitted to our hospital (Fig. 1).

On admission, his blood pressure was 132/86 mmHg; pulse 78/m; temperature 37.6°C; skin and costoabdominal findings were physically normal. He was alert but depressed. He was disoriented with regard to time and place. Immediate, recent, and remote memory disturbances were observed. His rating on the Hasegawa dementia scale-revised (HDS-R) was 12 (30); his performance on the Wechsler adult intelligence scale-revised (WAIS-R) was verbal IQ 56, practice IQ 49, total IQ 47. Aphasia, apraxia, and agraphia were not noted. Pupils were round and equal, and reaction to light was prompt on both sides. Deep tendon reflexes were slightly increased, but Babinski sign was negative. No cerebellar, sensory, or urinary disturbance was observed. Nuchal stiffness was negative.

Laboratory data

Peripheral blood count and blood chemistry values were normal; carcinoembryonic antigen (CEA) 2.4 ng/ml, neuron specific enolase (NSE) 8.4 ng/ml, and anti-Hu and anti-Yo antibodies negative; chest X-ray and ECG were normal. The serum Venereal Disease Research Laboratory (VDRL) test was 16x, and Treponema pallidum hemagglutination (TPHA) 20,480x. Herpes simplex virus (HSV) complement fixation test was 16x, and HSV IgG enzyme immunoassay (EIA) 83.5 U. Human immunodeficiency virus (HIV) was negative, as was human T-lymphotropic virus type I (HTLV-I). Brain CT showed mild cerebral atrophy, predominantly in the bilateral frontotemporal lobes. MRI T-2 weighted images revealed high signal lesions in both medial temporal lobes (Fig. 2A). MRI proton images exhibited more clearly the lesions in both limbic systems, with a few small lesions in the left caudate nucleus and in the right lateral thalamus (Fig. 2B, C). Magnetic resonance angiography (MRA) showed no narrowing or stenosis of cerebral arteries. An EEG revealed slow waves without par-
Clinical course

The patient was orally given Penicillin G (PCG) at 1.2 million units for 10 days, and this regimen was administered twice thereafter. The patient’s score on the dementia scale improved slightly, and the MRI signal abnormalities had decreased at the one-month follow-up (Fig. 2D).

Discussion

This patient had two episodes of TGA-like attacks consisting of transient disorientation and memory disturbance. In this case, the patient did not return to the normal mental state, which is atypical of cases of TGA attacks (5). Following the second TGA-like attack, our patient exhibited moderate dementia with CSF pleocytosis and a high syphilitic antibody titer. MRI showed abnormal signals in the bilateral amygdala and hippocampal regions, including a few lesions in the basal ganglia and thalamus, and also mild cortical atrophy. Although meningovascular involvement might have played a role in this case, parenchymatous inflammation as seen in general paresis appears to have been the major pathological process, giving rise to the neuroimaging and clinical presentations.

With regard to the CT or MRI findings of meningovascular syphilis, Holland et al (3) presented multifocal infarctions correlated with varying degrees of narrowing and ectasia of the supratentorial cerebral artery. However, in a review of the current literature, limbic lesions have not been described in syphilitic meningovasculitis (1, 3, 4). On the other hand, in general paresis, cerebral cortical atrophy and white matter changes have been emphasized (6, 7). In only one of 3 cases described by Zifko et al (7), high signal lesions and marked atrophy were observed in both hippocampi at the chronic stage.

Herpes simplex encephalitis, non-herpetic acute limbic encephalitis, and paraneoplastic limbic encephalitis often involve the limbic system (8–10). However, herpes simplex encephalitis was excluded because no HSV genome or antibody was found in the CSF. Non-herpetic limbic encephalitis is characterized by acute encephalitis and comparatively good outcome (9), but our case had a subacute onset and sustained with moderate dementia as a sequela. Paraneoplastic limbic encephalitis was also ruled out based on the lack of malignancies and anti-Hu and Yo antibodies.

It might be concluded that the present patient exhibited TGA-like symptoms as a manifestation of neurosyphilis, but it is difficult to clearly identify the underlying pathology; i.e., meningovasculitis of the small vessels of the limbic system, or general paresis with predominant limbic involvement. In addition, our report suggests that neurosyphilis should be considered when young patients present with a TGA-like attack and/or dementia.
Neurosyphilis with TGA-like Attacks

Figure 2. A) Axial T2-weighted image (1.5 T superconducting magnet system, SE 2,500/100) reveals hyperintense lesions in bilateral amygdala and hippocampi, predominantly on the right side. B) Axial proton image (SE 2,500/20) more clearly demonstrates similar lesions in the same regions. C) Axial proton image (SE 2,500/20) presents a few small lesions in the left caudate nucleus (right arrow) and right lateral-medial thalamus (left arrow). D) The MRI abnormalities (SE 2,500/100) have decreased one month later.
References