Central nervous system tuberculosis masquerading as primary dementia: a case report

Gruźlica ośrodkowego układu nerwowego naśladująca pierwotne otępienie: opis przypadku

Nitin K. Sethi[1], Prahlad K. Sethi[2], Josh Torgovnick[3], Edward Arsura[4]

[1]Department of Neurology, New York-Presbyterian Hospital, Weill Cornell Medical Center, New York, NY, U.S.A.
[2]Department of Neurology, Sir Ganga Ram Hospital, New Delhi, India
[3]Department of Neurology, Saint Vincent's Hospital and Medical Centers, New York, NY, U.S.A.
[4]Department of Medicine, Saint Vincent's Hospital and Medical Centers, New York, NY, U.S.A.

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Abstract

Primary dementias are the most common cause of memory impairment in patients above the age of 60. Hypothyroidism, depression, vitamin B_{12} deficiency and infectious diseases such as syphilis at times may present with memory impairment mimicking primary dementias in their clinical presentation. We present here a 64-year-old female who presented with complaints of forgetfulness, confusion, memory loss and impaired concentration for the past 3 months. Neuroimaging and computed tomography of the chest were suggestive of active tuberculosis. Anti-tubercular therapy led to resolution of enhancing lesions in the brain and abatement of memory deficits.

Key words: dementia, Alzheimer's disease, infection, tuberculoma, CNS tuberculosis.

Streszczenie

Otępienia pierwotne są najczęstszą przyczyną zaburzeń pamięci u pacjentów powyżej 60. roku życia. Niedoczynność tarczycy, depresja, niedobór witaminy B_{12} i choroby zakaźne, np. kiła, mogą niekiedy manifestować się zaburzeniami pamięci, które naśladują obraz kliniczny otępienia pierwotnego. W pracy przedstawiono przypadek 64-letniej kobiety, która zgłosiła się z powodu utrzymujących się od 3 miesięcy zapominania, zmącenia, utraty pamięci i upośledzonego skupienia uwagi. Badania obrazowe układu nerwowego i tomografia komputerowa klatki piersiowej wskazywały na aktywną gruźlicę. Leczenie przeciwprątkowe doprowadziło do wycofania wzmacniających się zmian w mózgu i ustąpienia zaburzeń pamięci.

Słowa kluczowe: otępienie, choroba Alzheimera, zakażenie, gruźliczak, gruźlica OUN.

Introduction

Central nervous system infectious diseases can at times mimic primary dementias in their clinical presentation. It is imperative that they be timely identified as appropriate treatment frequently has a gratifying outcome.

Case report

A 64-year-old woman working in a consulate presented with complaints of forgetfulness, loss of memory and confusion for the past 3 months. She was progressively experiencing difficulty in remembering and recalling important telephone numbers. Soon she was un-

Correspondence address: Nitin K. Sethi, MD, Comprehensive Epilepsy Center, New York-Presbyterian Hospital, Weill Cornell Medical Center, 525 East, 68th Street, New York, NY 10065, phone +212-746-2346, fax +212-746-8845, e-mail: sethinitinmd@hotmail.com
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able to handle the tasks of her daily work, increasingly depending upon her subordinates. She was a non-smoker and had no microvascular or macrovascular risk factors, namely hypertension, diabetes or coronary artery disease. There was no prior history of tuberculosis.

Examination revealed an afebrile normotensive woman disoriented to place and time. She was unable to perform simple arithmetic tasks, had constructional apraxia and scored 18/30 on the Mini-Mental State Examination (MMSE). Right-sided homonymous hemianopia was noted, though there was no papilloedema. Motor examination was normal except for mild incoordination noted on the right finger to nose test. Taking into consideration her age and the predominance of cognitive deficits with paucity of motor and cranial nerve signs on presentation, a presumptive diagnosis of Alzheimer's dementia was entertained even though the duration of symptomatology was less than 6 months.

Magnetic resonance imaging (MRI) of the brain revealed ring enhancing solitary lesions in the left frontal lobe and conglomerate ring enhancing masses in the left and right temporo-occipital region with surrounding vasogenic edema (Fig. 1). Contrast-enhanced computed tomography of the chest revealed multiple necrotic lymph nodes in the paratracheal region (Fig. 2). Blood tests revealed no evidence of an immunodeficiency state. Serum angiotensin-converting enzyme (ACE) level was normal. Repeated sputum cultures came back negative for acid fast bacilli. Mantoux tuberculin skin test was positive with induration of 7 mm. Cerebrospinal fluid (CSF) examination was deferred for fear of precipitating herniation due to mass effect in the brain. Since tuberculosis is endemic in India, the presence of multiple intracranial lesions along with multiple necrotic lymph nodes in the chest was felt to be highly suggestive of disseminated tuberculosis. She was treated with anti-tubercular therapy for one year along with a tapering course of steroids. Brain MRI one year later showed near complete resolution of the ring enhancing lesions with resolution of the surrounding vasogenic edema (Fig. 3). The patient was able to return to her work fully functional with no residual memory deficits.

Discussion

Primary dementias such as Alzheimer's disease constitute the most common cause of memory impairments and cognitive deficits in the elderly population. Depression (pseudodementia), vitamin B_{12} deficiency and

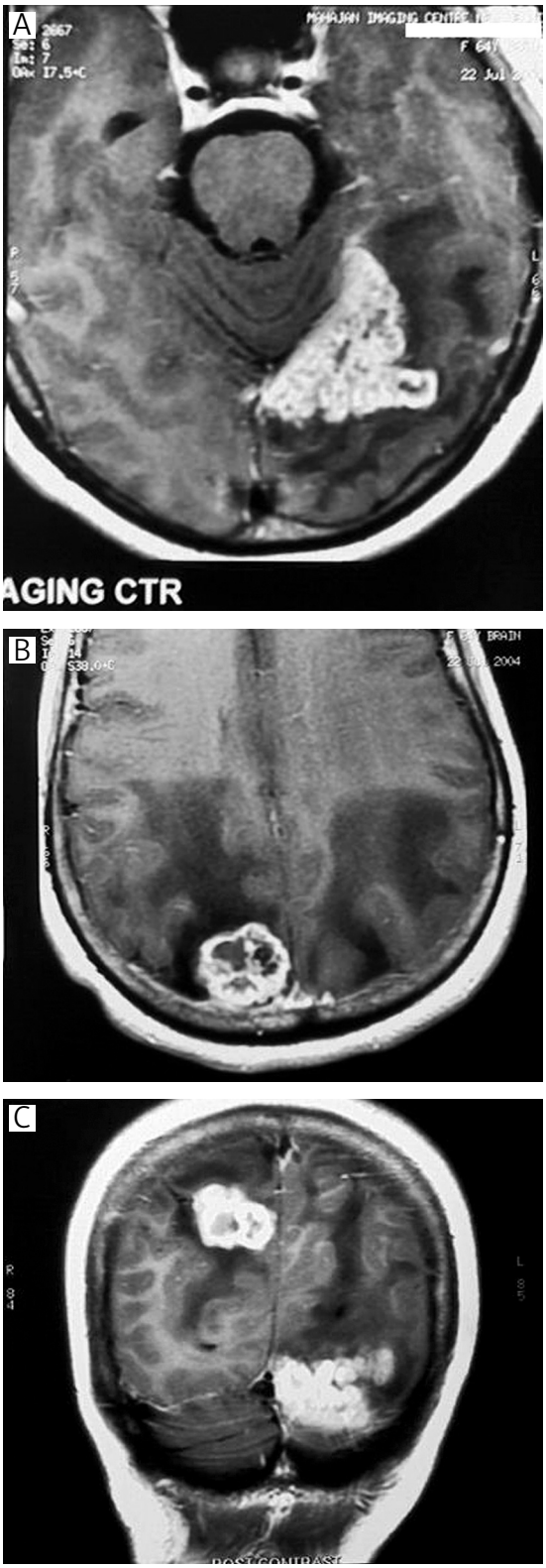

Fig. 1A-C. MRI of the brain showing ring enhancing solitary lesions in the left frontal lobe and conglomerate ring enhancing masses in the left and right temporooccipital region with surrounding vasogenic edema

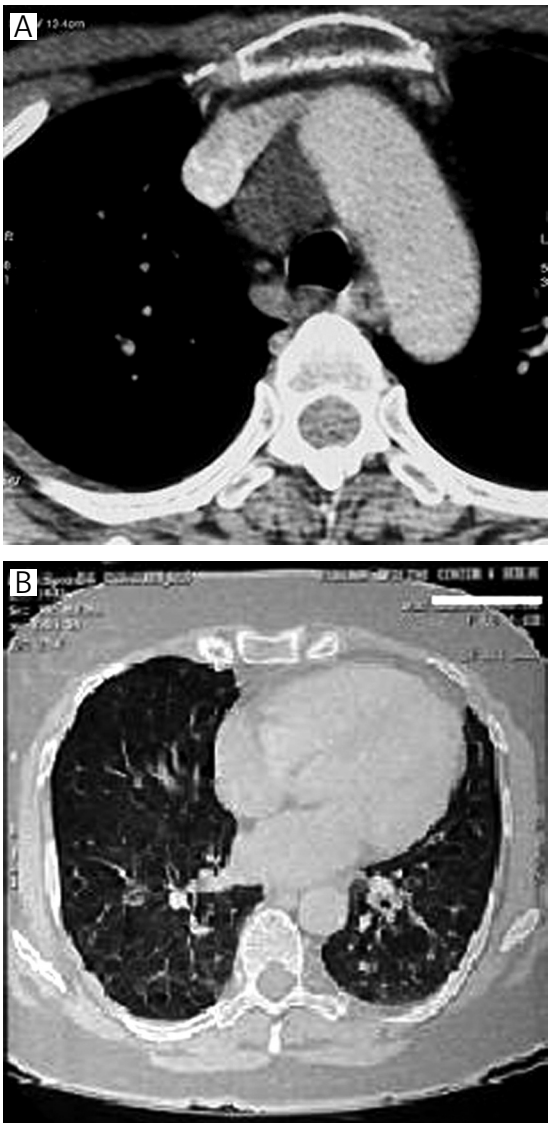

Fig. 2A-B. Contrast-enhanced computed tomography of the chest revealed multiple necrotic lymph nodes in the paratracheal region

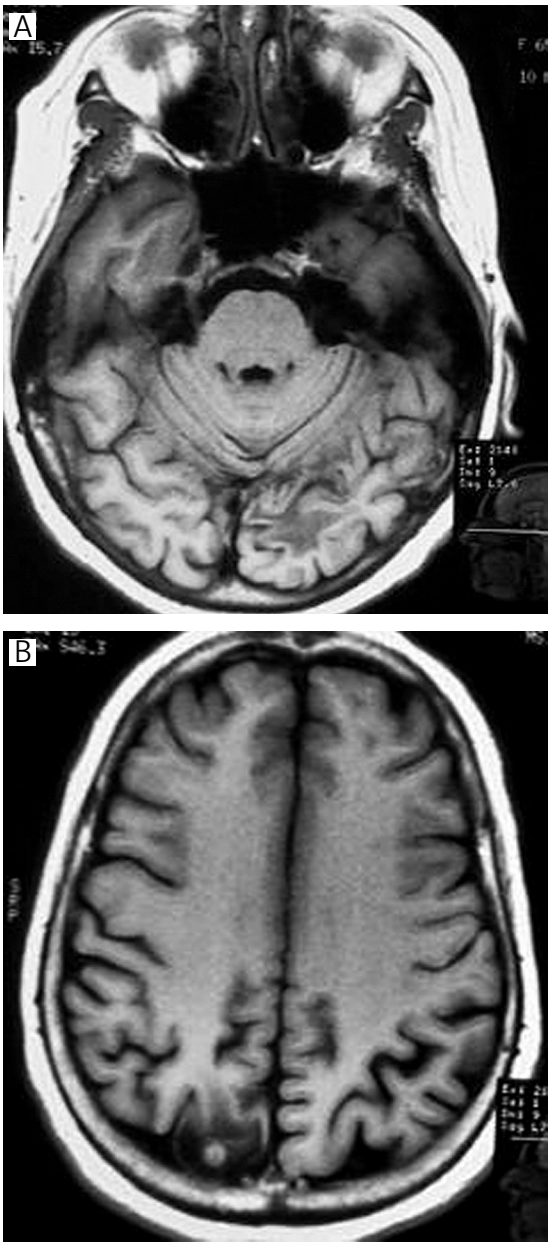

Fig. 3A-B. Brain MRI one year later showing near complete resolution of the ring enhancing lesions with disappearance of the surrounding vasogenic edema

hypothyroidism are listed as common reversible causes of memory impairment. Central nervous system infections can at times have a clinical presentation which mimics primary dementia. Schielke *et al.* presented the case of a 63-year-old male with an 8-month history of progressive cognitive decline. Like our patient, their patient too manifested signs of cortical dementia, namely verbal perseveration, non-fluent speech and constructional apraxia. Diagnostic work-up including an open frontal brain biopsy confirmed the diagnosis of neurosarcoidosis. Treatment with prednisolone resulted in a dramatic improvement with the patient becoming independent in activities of daily living [1]. Sundar *et al.* in their series of 76 patients with age less than 65 years found that 26/76 (34.21%) had a reversible cause of dementia. This reversible group comprised infections in 11/76 (14.47%), metabolic nutritional causes in 11/76 (14.47%) and autoimmune diseases in 3/76 (3.94%). In the infectious group, 3 patients had tubercular meningitis with imaging findings of basal meningitis in one and communicating hydrocephalus in two. Two patients had multiple tuberculomas, three patients had neurocysticercosis and in another three patients the cause was HIV (toxoplasmosis

in two and AIDS encephalopathy in one). A large proportion of their reversible cases were characterized by subcortical dementia [2]. Srikanth *et al.* in their series of 129 patients found reversible causes of dementia in 24 (18%) of the cases. Eleven of their patients had neuroinfections, 8 patients had normal pressure hydrocephalus while 5 had vitamin B_{12} deficiency. Neurosyphilis, cryptococcal meningitis, tuberculous meningitis, neurocysticercosis and HIV dementia were the reported etiologies in the infection group [3].

Our patient presented with cognitive deficits involving multiple domains, namely short-term memory, procedural memory, attention and calculation. She also exhibited constructional apraxia. Her clinical presentation hence was initially considered compatible with a cortical dementia syndrome. Neuroimaging and CECT chest in our patient raised the differential of active tuberculosis versus neurosarcoidosis. Though spinal taper was deferred, serum ACE level was not elevated. It is a well-established fact that patients with active tuberculosis may have negative sputum smear results. Further tuberculin skin test is of limited value in a country like India where tuberculosis is endemic and most of the population is BCG vaccinated at birth. In our patient we used a short 3-week tapering course of oral dexamethasone in conjunction with standard four-drug (isoniazid, rifampicin, ethambutol and pyrazinamide) anti-tubercular therapy for the initial two months followed by isoniazid and rifampicin in recommended doses. Since neurosarcoidosis is steroid responsive, we cannot rule it out completely. In our patient the response to anti-tubercular therapy after one year was extremely gratifying, with resolution of intracranial lesions and memory deficits. It was recommended that she continue anti-tubercular therapy for another 6 months for a total of 18 months.

Our case helps highlight a few points. Tuberculosis is endemic in India and can have myriad presentations [4]. Central nervous system tuberculosis in its initial stage may present with predominantly cognitive deficits mimicking dementia in its clinical presentation with a paucity of motor and cranial nerve signs. In a country like India where tuberculosis is rampant, central nervous system tuberculosis should be included in the differential diagnosis of a primary dementia syndrome.

Disclosure

The authors report no conflict of interest.

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