Never too old

Ricardo Krause Martinez de Souza, Rodrigo Tomazini Martins, Samanta Fabrício Blattes da Rocha, Henry Koiti Sato, Pedro André Kowacs

Lancet 2011; 378: 1676

Neurology Unit
(P A Kowacs MD, R K Martinez de Souza MD, R T Martins MD, H K Sato MD), **and Neuropsychology Unit, Instituto de Neurologia de Curitiba, Brazil**
(S F B da Rocha MSC)

Correspondence to:
Dr Pedro André Kowacs, Serviço de Neurologia, Instituto de Neurologia de Curitaba, Rua Jeremias Maciel Perretto 300, 80210-310 Curitaba, Brazil
pkowacs@gmail.com

In March, 2010, a 75-year-old woman was referred to us because of a 6-month history of progressive dementia. Her illness started with gait apraxia and progressed to cognitive decline with variable degrees of sphincter incontinence. She was an otherwise healthy woman with no history of previous medical conditions, and a widow of 15 years since her husband had died from a myocardial infarction. She had had three uneventful pregnancies, had never undergone surgery or blood transfusions, and did not come from a region with high HIV prevalence. She had loss of initiative and spontaneous speech, apathy, inattention, and short-term memory decline, and was unable to perform daily life activities without help. Neurological examination revealed an apraxic gait with small steps and a shift to the right, as well as a festinating gait. Her score on the mini-mental examination (MMSE) scale was 18. Neuropsychological examination showed impaired short-term memory, attention, and visuospatial abilities, and decreased mental processing speed. An electroencephalogram showed general slowing of background activity. Axial FLAIR MRI showed ventricular enlargement out of proportion to sulcal atrophy, and rounding of the frontal horns associated with periventricular hyperintensities, which suggested transependymal flow of cerebrospinal fluid (CSF) (figure). To exclude secondary dementia due to normal-pressure hydrocephalus,[1] we did a tap test,[2] which revealed normal opening pressure (17 mm Hg), with no improvement in gait or cognition. CSF examination showed leucocytosis (33 cells per µL, 91% lymphocytes and 9% neutrophils), high protein concentration (0·76 g/L), and normal glucose (2·27 mmol/L). Laboratory tests showed only an increased ESR (85 mm/h). CT of the chest was unremarkable. CSF PCR for tuberculosis, VDRL, and cultures and staining with China ink to detect fungi were negative. Serological tests for toxoplasma, cytomegalovirus, Epstein-Barr virus, herpes virus, and varicella zoster virus were negative. Treponemal antibody test (FTA-ABS) was also negative, but ELISA for HIV-1 and HIV-2 was positive and confirmed by western-blot analysis. CD4-cell count was 113 per µL. A diagnosis of HIV-associated dementia[3] was made. After 2 months on HAART, our patient's dementia reversed to mild cognitive impairment (MMSE score 28), and her urinary incontinence and gait apraxia improved. At final follow-up on Nov 8, 2010, she was in a stable clinical condition.

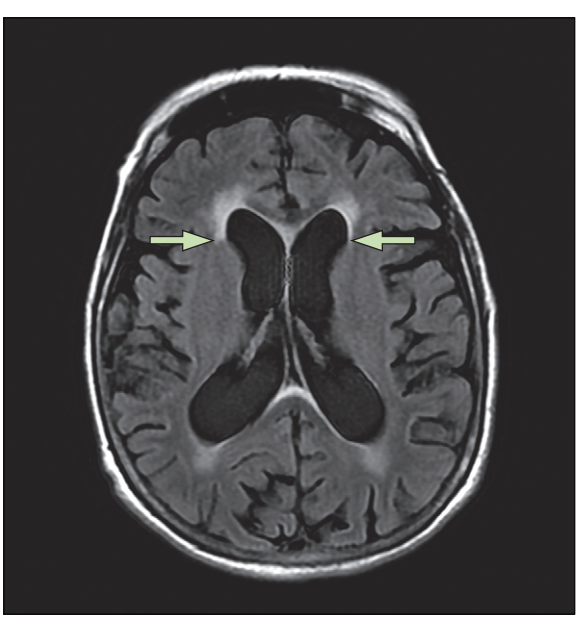

Figure: **Axial FLAIR MRI of head** showing enlarged and rounded ventricles associated with periventricular hyperintensities (arrows).

Our patient presented with clinical findings resembling classic and treatable Hakim-Adams syndrome (gait apraxia, dementia, and sphincter incontinence)[1] with compatible neuroimaging findings.[2] A possible mechanism for the pathophysiology of hydrocephalus is an inflammatory and toxic process in the CSF (an increase in the number of cells and the protein concentration) caused by HIV. HIV-related dementia generally occurs late in the course of AIDS in younger patients,[3] but it is not common as a presenting symptom in elderly patients.[4] However, the life expectancy and sexual activity of this population have increased,[5] and HIV infection has become treatable. Our patient improved with HAART and without shunting. HIV infection should therefore be considered as a possible diagnosis and HIV testing done even in older individuals presenting with any dementia syndrome.

References

1 Adams RD, Fisher CM, Hakim S, Ojemann RG, Sweet WH. Symptomatic occult hydrocephalus with "normal" cerebrospinal-fluid pressure: a treatable syndrome. *N Engl J Med* 1965; **273:** 117–26.

2 Marmarou A, Bergsneider M, Klinge P, Relkin N, Black PM. The value of supplemental prognostic tests for the preoperative assessment of idiopathic normal-pressure hydrocephalus. *Neurosurgery* 2005; **57** (3 suppl): S17–28.

3 McArthur JC, Hoover DR, Bacellar H, et al. Dementia in AIDS patients: incidence and risk factors. Multicenter AIDS Cohort Study. *Neurology* 1993; **43:** 2245–52.

4 McMurtray A, Clark DG, Christine D, Mendez MF. Early-onset dementia: frequency and causes compared to late-onset dementia. *Dement Geriatr Cogn Disord* 2006; **21:** 59–64.

5 Lindau ST, Gavrilova N. Sex, health, and years of sexually active life gained due to good health: evidence from two US population based cross sectional surveys of ageing. *BMJ* 2010; **340:** c810.