

CNS toxoplasmosis induced hydrocephalus revisited and a brief review of AIDS dementia complex

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Dear editor:

I read with interest the article "CNS toxoplasmosis presenting with obstructive hydrocephalus in patient of retroviral disease – a case series"¹ by A Basavaprabhu al, published in the Medical Journal of Malaysia volume 67.

Unfortunately I found obvious error and misinterpretation of the presented images. In figure 1 of the article, the authors had wrongly arrowed the 3rd ventricle as 4th ventricle.

The figures 1 and 2 show diffuse prominent cerebral sulci which together with the dilated ventricular system are suggestive of underlying cerebral atrophy rather than hydrocephalus. The cerebral atrophy is disproportionate to the patients' age but the appearances are commonly seen in patients with low CD4 counts complicated with AIDS dementia complex (ADC). The additional finding of abnormal hypoattenuation in the periventricular and deep white matter on both figures can also be related to ADC which is known to cause demyelination and gliosis of the white matter².

The diagnosis of ADC is based on clinical neurological findings rather than a radiological diagnosis. Hence, I would suggest the authors to consider this entity with further clinical correlation.

REFERENCES

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2. Smith AB, Smirniotopoulos JG, Rushing EJ. From the archives of the AFIP: central nervous system infections associated with human immunodeficiency virus infection: radiologic-pathologic correlation. Radiographics 2008; 28: 2033-58.

Reply:

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We thank MB Chow for his interest in our article. We would like to clarify two points.

We agree with him that we have labeled the third ventricle as the fourth ventricle.

The error is regretted.

However, the AIDS dementia complex(ADC), also referred to as HIV-associated dementia (HAD), is the most severe form of HIV-associated neurocognitive disorders (HANDs). HAD is associated with marked cognitive impairment involving at least two cognitive domains and the cognitive impairment produces marked interference with day to day functioning¹. Our patient (case1) presented with acute onset altered sensorium, had high levels of antitoxoplasma IgG and he showed excellent response to antitoxoplasma treatment. So we made a diagnosis of CNS toxoplasmosis in our patient. CNS toxoplasmosis can present as ventriculitis and obstructive hydrocephalus without evidence of focal parenchymal lesions in HIV patients². We did not do a repeat imaging after treatment as there was no residual neurological deficit and our patient also had financial problems. As our patient had an acute onset neurological deficit from which he recovered completely (as mentioned in our case series) we did not consider a diagnosis of HAD. Considering a diagnosis of HAD based solely on radiological findings in a patient with an acute onset neurological deficit which has improved completely in the absence of neuropsychological testing is inappropriate.

CT scan, Serology, CSF Picture, Low CD4 counts and response to anti toxoplasma therapy in our patient suggested Toxoplasma encephalitis as per the Centre for Disease Control (CDC) case definition for toxoplasma encephalitis.

REFERENCES

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This article was accepted: 13 March 2013

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