CASE REPORT

CNS Toxoplasmosis Presenting with Obstructive Hydrocephalus in Patients of Retroviral Disease-A Case Series

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SUMMARY

CNS toxoplasmosis presenting as hydrocephalus is a very rare entity. We present three cases of HIV positive patients whose brain imaging revealed hydrocephalus and who improved with anti toxoplasma medication along with intravenous steroids and did not require any CSF shunting procedures. The mechanism of hydrocephalus in CNS toxoplasmosis is usually due to compression of CSF outflow pathway by ring enhancing lesions but even in their absence hydrocephalus can be rarely seen due to ventriculitis. Hence in HIV positive patients with unexplained hydrocephalus CNS toxoplasmosis should be considered and such patients if started on treatment early have a good prognosis without requiring neurosurgical intervention.

KEY WORDS:
CNS toxoplasmosis; HIV; hydrocephalus; ventriculitis

INTRODUCTION

The manifestation of CNS toxoplasmosis as obstructive hydrocephalus in adults is rare and only few cases have been reported worldwide. We present three cases of CNS toxoplasmosis with obstructive hydrocephalus seen in patients with retroviral disease which were all treated medically with anti toxoplasma drugs and improved without any surgical intervention.

CASE REPORT

Case 1:-

28 year old male, diagnosed to have retroviral disease 2 years back, on HAART (Highly Active Anti Retroviral Therapy) since 1 year presented with fever for three days, generalised tonic clonic seizures and decreased level of consciousness since last two days. On examination patient was unconscious with GCS of 5/15. His blood investigation showed hyponatremia (sodium 121meq/l), CD4 count was 125. Contrast enhanced computerised tomogram (CECT) head showed multifocal non-enhancing hypodense areas in bilateral lentiform nucleus, right corona radiata, bilateral parietal lobes with obstructive communicating hydrocephalus (figure 1). Patient was started on steroids and mannitol and lumbar puncture was done for CSF analysis which showed protein of 164 mg/dl, glucose 61 mg/dl, cell count of 25 cells/cumm with 97% lymphocytes and 3% neutrophils. CSF analysis was negative for India ink stain for Cryptococcus, cryptococcal antigen, acid fast bacilli and VDRL. Serum IgG toxoplasma was positive with titre of 226.694 IU/ml. Patient was treated with anti toxoplasma drugs. Within 7 days of treatment there was significant improvement in patient's condition. Steroids and mannitol were tapered and patient was discharged on anti toxoplasma drugs for a period of three weeks followed by secondary prophylaxis with sulfamethoxazole/ trimethoprim. Patient is now on regular follow up.

Case 2:-

29 year old male, diagnosed with retroviral disease 7 years back on HAART and co- trimoxazole prophylaxis (CD4 four months back 112cells/cumm) was admitted with complaints of vomiting since two days, seizures since one day followed by loss of consciousness. On examination patient was unconscious with GCS OF 6/15. His blood investigation showed hyponatremia (sodium 128 meq/L) and CD4 count of 73/cumm. CECT head showed multiple ring enhancing lesions in bilateral frontal, basal ganglia, thalamus and right cerebellar region adjacent to fourth ventricle with obstructive hydrocephalus. Patient was started on dexamethasone and mannitol and CSF analysis showed protein of 246.8 mg/dl, glucose of 44 mg/dl, 20 cells with 84% lymphocytes, 8% neutrophils. CSF India Ink, AFB stain, VDRL and culture were negative. Serum IgG toxoplasma was positive with titre of 210.099 IU/ml. Patient was treated with anti toxoplasma drugs. Within one week patient was fully conscious without any residual neurological deficit and was then discharged and is now on regular follow up.

Case 3:-

40 year old male patient diagnosed of retroviral disease five years back, on HAART since last five months (CD4 at ART initiation was 22/cumm) presented with complaints of headache and vomiting since one week. Neurological examination was normal.CECT head showed ischemic changes in bilateral corona radiata, centrum semiovale and periventricular regions with obstructive hydrocephalus (figure 2). Investigation showed CD4 cell count 107/cumm and serum IgG toxoplasma titre of 125.5 IU/ml.Since there

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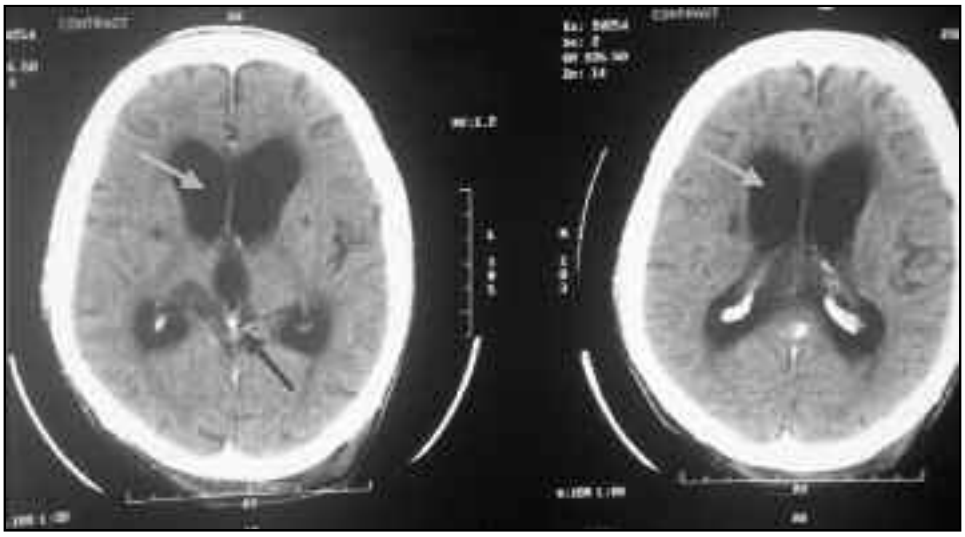

Fig. 1: Contrast CT brain of the patient shows hydrocephalus. White arrow shows dilated lateral ventricles and red arrow shows prominent fourth ventricle.

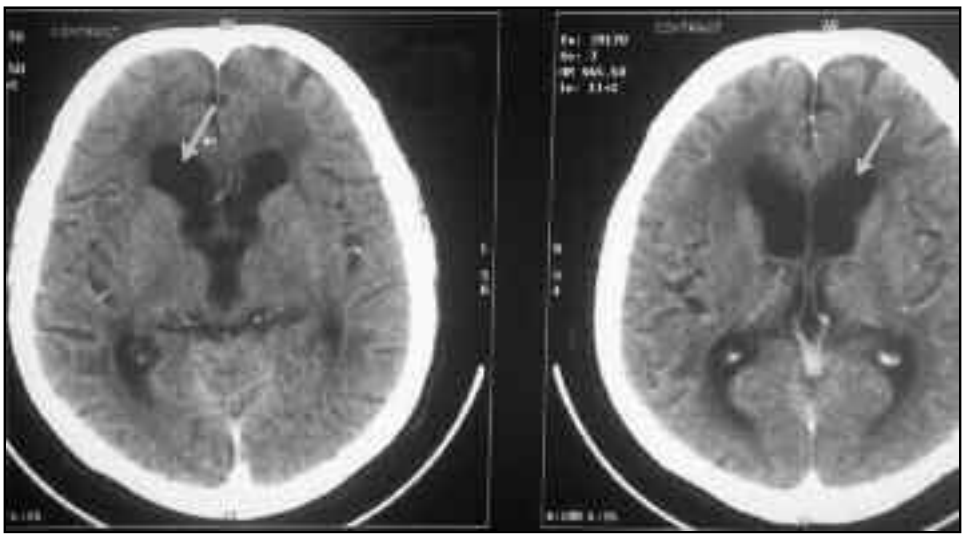

Fig. 2: Contrast CT of the patient showing dilated lateral ventricles.

was significant improvement in CD4 count after ART initiation a possibility of IRIS (Immune Reconstitution Inflammatory Syndrome) was considered. CSF analysis showed protein of 158.2mg/dl, glucose 29mg/dl, cells 50/cumm with 78% neutrophils, 19% lymphocytes. CSF was negative for India ink stain, AFB stain, VDRL and culture. Patient was treated with anti toxoplasma drugs along with steroids and mannitol. On fourth day onwards patient's headache improved. He was then discharged and is on regular follow up.

DISCUSSION

Toxoplasmosis is the most common CNS opportunistic infection seen in HIV positive patients[1]. CNS toxoplasmosis remains a highly prevalent disorder of the central nervous system even in the era of HAART, particularly among severely immunosuppressed patients and in the absence of prophylaxis[2]. Most common presentations are of single or multiple ring enhancing lesions, which are generally localised at corticomedullary junction, in white matter or basal ganglia or of a diffuse cerebritis[3]. Hydrocephalus due to cerebral toxoplasmosis is very rare and only few cases have been reported in the literature. The mechanism of hydrocephalus is compression of CSF pathway by surrounding parenchymatous space occupying lesions or a necrotising ependymitis and plexitis obstructing CSF flow. Though tubercular meningitis and cryptococcal meningitis are more common causes for obstructive hydrocephalus, CNS toxoplasmosis should be ruled out in obstructive

hydrocephalus of unknown origin. It is difficult even for the best of clinicians to distinguish CNS Toxoplasmosis from CNS Tuberculosis when Toxoplasmosis presents with obstructive hydrocephalus in the absence of parenchymal lesion. All 3 cases in our report had negative CSF Analysis results for Mycobacterium Tuberculosis and Cryptococcus neoformans.

MRI scan is more accurate than CT scan to detect lesions of CNS toxoplasmosis[4]. Postcontrast periventricular enhancement on imaging is consistent with ventriculitis[5].

Neuropathological examination will show infiltration with lymphocytes, macrophages and plasma cells, microglial nodules and hyalinization and tachyzoites of Toxoplasma gondii[5]. Although positive brain biopsy is the ideal investigation but because of its invasiveness, CNS imaging and serology can be substituted for diagnosis and regression of lesions and clinical improvement with empirical anti toxoplasma treatment gives a definite clue. In our case series brain biopsy and repeat brain imaging after completion of therapy to demonstrate resolution of hydrocephalus were not done for any of the patients due to financial constraints.

All 3 cases improved with medical management. All three patients were treated with sulfamethoxazole/trimethoprim (800mg/160mg thrice daily) and sulfadoxine/pyrimethamine (500 mg/25mg thrice daily) along with folic acid 5mg daily. Permanent CSF shunting may not be necessary for hydrocephalus due to CNS toxoplasmosis and external CSF shunting may only rarely be needed.

CONCLUSION

Our case series highlights the fact that CNS Toxoplasmosis can present with obstructive hydrocephalus which responds to medical line of management and neurosurgical intervention is rarely required.

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