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HEMIPLEGIA IN A CASE OF HIV

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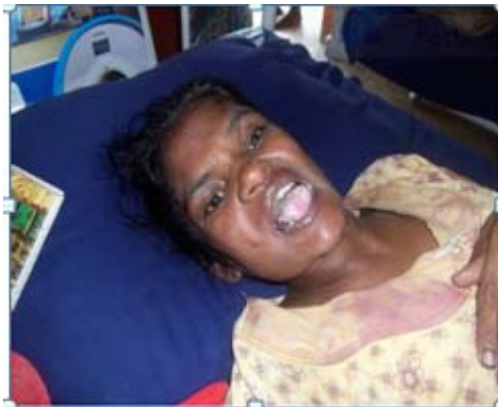
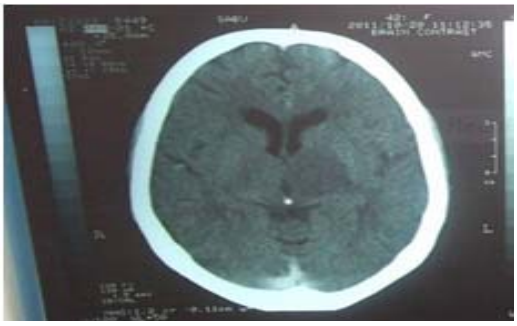
Abstract: Toxoplasmosis is caused by a parasite Toxoplasma gondii.Toxoplasmosis is a principal opportunistic infection of the central nervous system in AIDS. 33 percentage of patients present with seizures and 60 percentage of patients present with focal neurological deficit. Infection occurs when CD4 count is less than 200 micro litre. Here, we present a case of cerebral toxoplasmosis who presented with focal seizures and hemiplegia.
Keyword :TOXOPLASMOSIS,SEIZURES,HEMIPLEGIA.

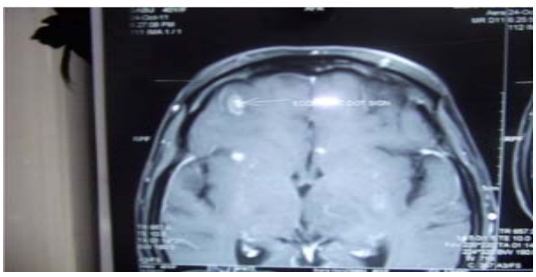
A 47yrs old female was admitted in our hospital with a history of one episode of seizure involving right upperlimb (UL) and lowerlimb (LL),which lasted for few minutes following which she was found to have difficulty in using the right UL and LL .No significant past history was elicitable and she gave H/o extra marital contact. General examination was found to be normal except for pallor and oral candidiasis (fig 1).CNS examination showed a weakness of right UL and LL with a power of grade 0 and 4 respectively, with no response in right plantar reflex Other systems examination were found to be normal. On 3rd day of admission right LL power worsened to grade 1. Routine investigations were found to be normal. CT brain with contrast showed a hypodense lesion in the left thalamus, internal capsule, left frontal white matter with minimal mass effect, without midline shift suggestive of probable granuloma(fig:2)

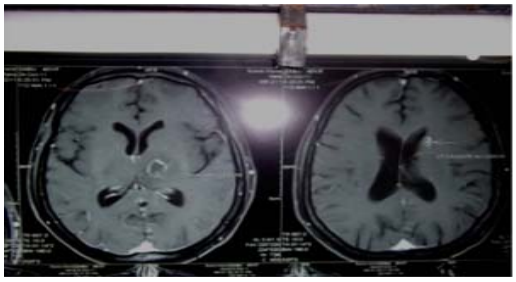




(fig1: oral cavity of the patient showing candidiasis)
(fig 2: computed Tomography of the patient showing hypodense lesions in left thalamus,internal capsule and left frontal white matter)

Test for HIV by ELISA was positive and CD4 count was 128/ µl .Toxoplasma IgG titre was 3.91 OD ratio(ref. range 1.11 positive) and IgM titre was 0.41 OD ratio.(ref.range 1.11 positive,<0.9 negative) CSF showed, sugar-60mg/dl, proteins-48mg/dl, leucocytes -2/ul, and there was no cobweb formation.Mantoux skin test and sputum for acid fast bacillus were negative.MRI brain showed multiple, varying size, ring enhancing lesion in T2 W image with eccentric dot(fig 3) noted in the left thalamus, precentral, right parietal ,frontal and left caudate nuclei(fig 4).Mild mass effect noted in the left thalamus (fig 4).On MRI spectroscopy there was a significant elevation of lactate peak and mild elevation of choline peak, suggestive of TOXOPLASMOSIS.





(fig:3, fig:4 Magnetic Resonance Imaging of the patient showing lesions in right frontal, left thalamus, left caudate nucleus with the characteristic "eccentric dot sign")

Final diagnosis of CNS TOXOPLASMOSIS with CATEGORY C HIV INFECTION was made ,which presented as HEMIPLEGIA .We started the patient on oral phenytoin 100mg OD, Cotrimoxazole DS BD,(trimethoprim 160mg and sulphamethoxazole 800mg),T. Fluconazole 150 mg OD and ART. ART (T.zidovudine300bd,T.Lamivudine 150mg bd,T.Nevirapine 200mg od) . Physiotherapy was also started .After 2 weeks of treatment power in right UL improved to grade 2 and LL improved grade 4. We discharged the patient with the advice to continue the drugs and to recheck CD4 count after two months .

DISCUSSION:

Toxoplasmosis is caused by infection with the obligate intracellular parasite *Toxoplasma gondii* . Active infection in the immunocompromised host is most likely to be due to the spontaneous release of encysted parasites that undergo rapid transformation into tachyzoites within the CNS .Transmission is through ingestion of sporulated oocysts from contaminated water ,soil, food or bradyzoites from under cooked meat. Other modes of transmission are blood or organ donation and transplacental transmission. 1

Infection with *T. gondii* induces humoral and cell-mediated immune responses. T cells, macrophages, and type 1 cytokines (IFN-, IL-12) are crucial for control of *T. gondii* infection.CD8+ T cells are primarily responsible for this resistance, although significant protection also is conferred by CD4+.The costimulatory molecules CD28 and CD40 ligand are pivotal for the regulation of IL -12 and IFN- production in response to the parasite2. More recent studies have shown
that expression of CD40L is defective on CD4+ T cells from HIV-infected patients.3 This deficiency may play a role in defective IL-12/IFN- production associated with HIV infection.

The most common manifestation of acute toxoplasmosis in immunocompetent individual is
cervical lymphadenopathy. 20 - 40% of patients presents with headache ,malaise ,fatigue and fever. Small proportion of patients have myalgia, sorethroat, abdominal pain , maculopapular rash , meningoencephalitis and confusion.1

In immunocompromised individual toxoplasmosis infection occurs when CD4<200ul.In >95% of cases it is due to recrudescent infection. More than 50% of patients with clinical manifestation have intracerebral involvement.Clinical findings may range from nonfocal to focal dysfunction .Patient may present with altered mental status (75%),fever(10-72%),seizures(33%),head ache(56%)and focal neurological finding(60%),cranial nerve palsies, movement disorder, dysmetria, visual field loss and aphasia.1

Other opportunistic infection which can present similarly are,primary CNS lymphoma, Progressive Multifocal Leucoencephalopathy, and Tuberculoma.Primary CNS lymphoma is usually subacute in onset, Confusion, lethargy and memory loss are the most frequent symptoms, CD 4 count is usually< 50/ul.CSF will show elevated protein count MRI will show solitary mass lesion which are as frequent as multiple lesion.Sub ependymal enhancement is more specific of CNS lymphoma.PML usually occurs when CD 4<200/ul.Presenting symptoms are limb weakness, altered mental status, gait ataxia and visual symptoms.MRI usually shows non enhancing lesion and they are not surrounded by edema and substantial mass effect on surrounding structure is absent. MRI picture of tuberculoma may have lesions with central necrosis shown as T2W hyperintense signal with surrounding peripheral hypointense rim.

Diagnosis of cerebral Toxoplasmosis is based on clinical presentation ,positive serology and radiological evaluation. More than 97% of patients with AIDS and toxoplasmosis have IgG antibody. IgM serum antibody is usually not detectable .Double contrast CT will show single and frequently multiple contrast enhanced lesion < 2cm.MRI usually demonstrates multiple lesion located in both hemisphere, basal ganglia and cortico medullary junction. Eccentric target sign is a classical finding in MRI. 1

The central enhancing core of the target seen on MRI was produced by a leash of inflamed vessels extending down the length of the sulcus that was surrounded by concentric zones of necrosis and a wall composed of histiocytes and proliferating blood vessels, with impaired permeability producing the peripheral enhancing rim.4 Pyrimethamine combined with sulfadiazine and folinic acid is the therapy of choice for AIDS patients with toxoplasmosis and is the standard to which experimental regimens should be compared. This regimen is associated with clinical response in 68% to 95% of patients with CNS Toxoplasmosis.5 TMP-SMX6 (at 10 mg/kg/day of the trimethoprim component divided in two doses) showed similar efficacy to the pyrimethamine-sulfadiazine regimen (with a more rapid radiologic response in the TMP-SMX group) in a randomized pilot trial in 77 patients with AIDS7

CONCLUSION:

All the cases presenting as hemiplegia are not due to vascular events. Particularly in immuno compromised individuals, we can think of opportunistic infections which can present as focal neurological deficits.

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