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SPECTRUM OF CENTRAL NERVOUS SYSTEM MANIFESTATIONS IN HIV/AIDS PATIENT

Radio-Diagnosis

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ABSTRACT

HIV is one of the viruses that belong to the Retroviridae family of retroviruses. Neurological problems, metastatic cancers, and immunodeficiency are associated with it. Since antiretroviral therapy (ART) was developed, the incidence of HIV-related central nervous system (CNS) illnesses has substantially decreased and the prognosis for those with HIV has improved tremendously. Even yet, a significant portion of people die from HIV-related CNS diseases. When it comes to early diagnosis and prompt treatment, advancements in magnetic resonance imaging (MRI) have improved the prognosis for those living with HIV. Central nervous system (CNS) symptoms of HIV encompass a wide range of diseases. Progression-related primary CNS lymphoma (PCNSL), HIV encephalopathy, progressive multifocal leukoencephalopathy (PML), CNS toxoplasmosis, and other opportunistic infections-related illnesses like CNS cryptococcosis, CNS tuberculosis, and immune reconstitution inflammatory syndrome (IRIS) are among them.

KEYWORDS

Central nervous system, HIV, Tuberculoma, CD4 count

INTRODUCTION

HIV is a neurotrophic, neuroinvasive, and neurovirulent pathogen [1]. It can infect the central nervous system (CNS) both directly and indirectly, but it also compromises T-cell-mediated immunity, making it more susceptible to several other neuro illnesses.[2-4] Prior to the development of combination antiretroviral therapy (cART), 10% of HIV-positive people are thought to have initially had neurological abnormalities, and 30–50% of them went on to develop neurological difficulties as their disease progressed.[5] CNS infections continue to be a major source of morbidity and mortality in the cART era, despite recent large observational cohorts of HIV-infected patients reporting a considerable drop in the overall incidence of the most common HIV-associated neurological illnesses. [6-8] HIV-positive patients may present with a wide range of clinical symptoms from a variety of pathogens, making the identification of CNS infections difficult because other illnesses need to be ruled out during the differential diagnosis.[9] A CD4 lymphocyte count of less than 200 has been found to be predisposing to cerebral toxoplasmosis, progressive multifocal leukoencephalopathy (PML), and cryptococcal meningitis.

This finding has been linked to the cause of CNS infections in HIV patients.[10,11] It is anticipated that patients with greater CD4 counts and HIV are infected with non-opportunistic infections.[9] Numerous research have shown the range of CNS infections in individuals with low CD4 count ranges [7,12–14], but the distribution of different CNS diseases in HIV-infected subgroups with greater CD4 count ranges has not been studied. Present study was conducted with the aim of to study spectrum of central nervous system findings in HIV/AIDS patients.

MATERIAL AND METHODS:

Study Design: Cross sectional descriptive study.
Study Site: Tertiary care teaching institute.
Study Timeline: 1 year
Sample size: 60

Inclusion Criteria: All the patients who had their HIV status confirmed via ELISA/ western blotting presenting with neurological symptoms and willing to give informed written consent to take part in the study.

Exclusion Criteria: Patients who were allergic to contrast media, those who had contra indications for MRI and those who were not willing were excluded from the study.

Data Collection: After approval from ethical committee and obtaining informed consent form, MRIs were performed on each patient who met the inclusion criteria.

Every magnetic resonance imaging test was conducted using a Siemens 1.5 Tesla magnetom avanto MR apparatus. There was a 0.5 mm space between each slice, and the slices were 4-5 mm thick. Gadolinium DTPA was injected intravenously (IV) at the prescribed dose of 0.1 mmol/kg or 0.2 ml/kg after the patient's history and information were taken.

The Following Sequences Were Obtained:
Pre-contrast

- Axial and coronal T1 weighted images
- Axial and sagittal T2 weighted images
- Axial fluid-attenuated inversion recovery (FLAIR)
- Diffusion weighted imaging (DWI)
- Susceptibility weighted imaging/gradientrecalled echo (SWI/GRE)

Post Contrast

- T1 weighted fat suppressed images.
- MP RAGE(3D T1 gradient echo sequence)
- Axial fluid - attenuated inversionrecovery (FLAIR)
- Additionally, MR-Spectroscopy, MR- Angiography /venography were done whenever required.

Statistical Analysis:
Quantitative data were represented in the form of values and percentages. Also the qualitative data was presented in the form of visual impression like bar- diagram, pie-diagram.

OBSERVATION AND RESULTS

Out of the 60 patients in our study, the largest number, or 20, (33.3%), belonged to the age range of 41–50 years, and followed by the age group of 31–40 years, which included 19 (31.67%). In both the 11–20 and over 60 age groups, there were the fewest patients—two. With a ratio of 1.86:1, the number of male cases exceeded that of female cases. Fever accounted with 41.67 percent of all clinical presentations, with altered sensorium (33.3%), headache (20%), stroke (3.33%), and dementia (1.67%) following in order of frequency.

In 63.33% of the patients, opportunistic infections were the most prevalent finding. 33.33% of patients experienced primary consequences of HIV, such as brain shrinkage and HIV encephalopathy. In 3.33% of cases, CNS lymphoma was detected. An infarct was seen in 6.67% of patients with cerebrovascular problems. Arachnoiditis and Pott's spine with para-vertebral abscesses were among the additional abnormalities observed in 5% (3/60) of the patients.Fig.-1) Tuberculoma T1 post contrast(A&B) showing multiple ring enhancing lesions in middle frontal gyrus and along ambent and quadrigeminal cisterns.

They are appearing T2 hypointense (C &D) with surrounding hyperintense areas.

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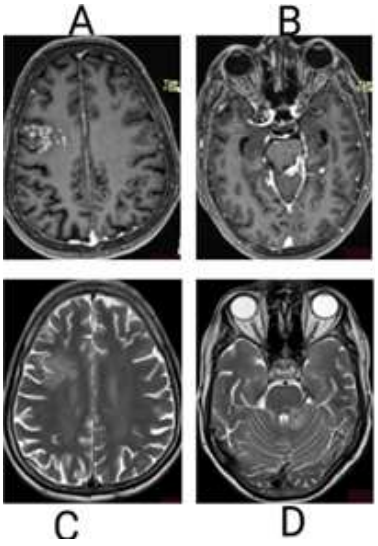

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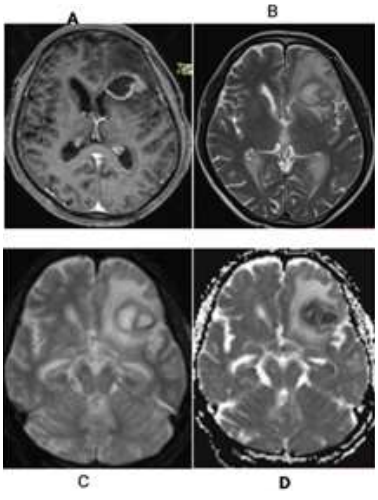

Fig. 2) CNS Lymphoma- T1post contrast (A) in immunocompromised patient showing well defined peripherally enhancing lesions in corpus callosum,right middle frontal gyri , left superior frontal gyrus,left corona radiate. They are appearing T2 (D) hyperintense showing diffusion restriction on DWI(B) with corresponding drop on ADC(C)

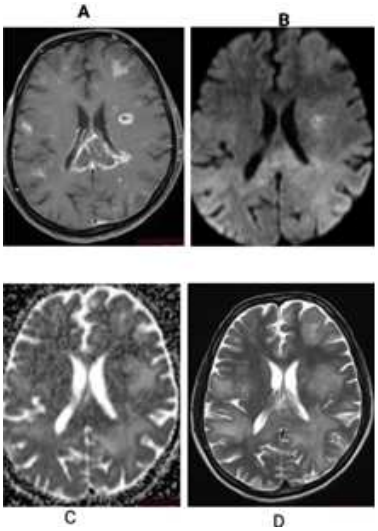

Fig. 3) Cerebral Abscess -T1 post contrast (A) peripheraly enhancing lesion appearing T2 hyperintense (B)with srronding hypointense rim noted in left frontal lobe showing marked peripheral edema with central area of diffusion restriction on DWI (C) and corresponding drop on ADC (D)

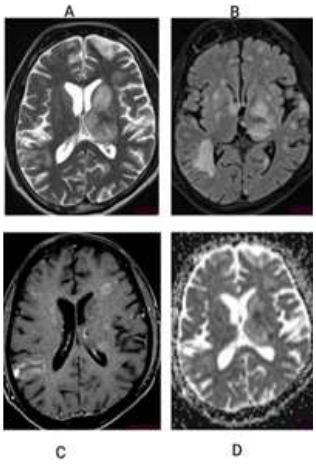

Fig. 4) CNS Toxoplasmosis -Multiple T2/FLAIR (A&B) hyperintense abnormal signal areas noted in bilateral cerebral hemisphere predominantly along basal gangliashowing no diffusion restriction and drop on ADC(D) and showing peripheral ring enhancement on T1 post contrast study(C)

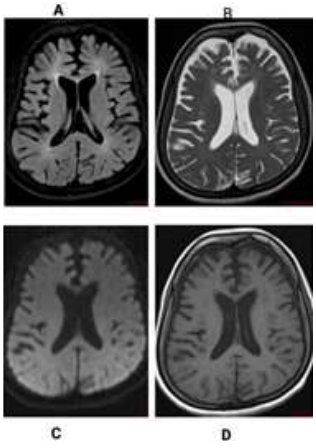

Fig. 5) HIV encephalopathy -20 yr old female patient presented with cognitive decline on FLAIR axial image (A) and T2(B) showing hyperintensity along bilateral deep white matter and periventricular region ,showing no diffusion restriction on DWI (C),and also appears iso to hypointense on T1 (D) with prominence of sulcal spaces due to marked cortical atrophy(age disproportionate)

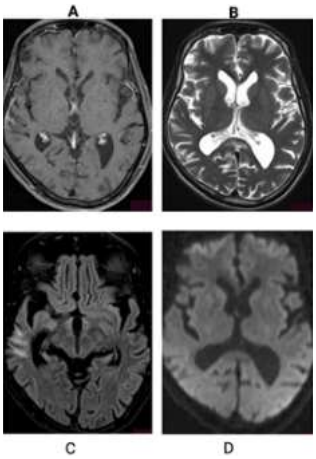

Fig. 6) Meningitis- FLAIR hyperintense areas (C) noted along sulcal spaces of right temporal lobe showing no diffusion restriction on DWI(D) , on T1 post contrast study (A)there is abnormal post contrast enhancement along sulcal spaces of right temporal lobe.CSF along sulcal spaces of right temporal lobe appears slightly hypointense on

T2(B)

Table 1: Diagnosis And Frequency Of Cns Pathologies In Hiv Patients

Pathologies	No. of patients	%
Primary effects of HIV	20	33.33
Cortical Atrophy	8	
HIV encephalopathy	12	
Opportunistic Infections	33	55
CNS Tuberculosis	20	
Toxoplasmosis	04	
Fungal Infections	02	
PML	07	
Lymphoma	02	3.33
Cerebrovascular complications	4	6.67
Infarction	4	
Hemorrhage	00	
Any other	1	1.67
Arachnoiditis	01	

In our investigation, isolated tuberculous meningitis was observed in 40% of individuals with CNS tuberculosis. Fifty-five percent of patients had isolated tuberculomas, and fifteen percent had tuberculous meningitis along with their tuberculomas. Ten percent of cases had a tubercular abscess.

Table 2: Spectrum of Neurotuberculosis in HIV patients

Imaging Diagnosis	No of cases	Percentage (%)
Tuberculoma	9	45
Tubercular meningitis	8	40
Tuberculomas with tubercular meningitis	3	15
Tubercular abscess	2	10
Total	20	100

Five cases of toxoplasmosis were found in our investigation. The gangliocapsular region was the most frequently found location, with multiple lesions present in 75% of individuals. In 75% of the patients, there was ring augmentation. Fifty percent of the cases had blooming on SWI. Eccentric target lesion-like enhancement was seen in two cases. Every patient displayed mass effect and surrounding edema. In every case, there were bilateral, asymmetric white matter lesions. The most frequent site was the subcortical white matter (86.67%), which was followed by the brain stem (50%). Diffusion restriction was uneven in one instance. There was no bulk impact or enhancement in any of the lesions. In 75% of instances, there was concomitant brain atrophy and ventricular prominence. There were two CNS lymphoma instances in our investigation. Every instance had more than one lesion. One of these two examples involved the supratentorium, which is white matter that is both periventricular and subcortical. On T1W images, every lesion was isointense. On T2W images, the lesions in one case were hypo-to isointense, whereas those in the other cases were hyperintense. Every example exhibited heterogeneous, ring-patterned, and homogeneous enhancement, with varying patterns of enhancement seen. There were two incidences with encircling oedema. There were 12 cases of HIV encephalopathy. Every single one of the twelve patients (100%) had bilateral symmetrical lesions. In 83.88% of cases, periventricular lesions were seen. There was no contrast enhancement in any of them. Meningitis was present in 18.33% of the 60 patients. CNS tuberculosis was linked to every episode of meningitis. In 6.67% of the patients in our investigation, an infarct-related imaging characteristic was present. In this specific cohort, HIV Vasculopathy was thought to be the diagnosis when other potential risk factors for CV stroke were ruled out and no clear etiological factors were found.

Table 3: CD4 Count and Neurological manifestations

Cd4 Level	TB	Cryptococcosis	Toxoplasmosis	PML	HIV Encephalopathy	Lymphoma
0-50	00	01	01	01	0	01
51-100	01	00	02	02	0	01
101-150	03	00	01	03	2	0
151-200	01	00	00	00	2	0
201-250	04	00	00	01	2	0
>250	11	00	00	00	6	0

DISCUSSION

When compared to other research conducted in India by HM Rana et al.[15], Sharma SR et al.[16], and S Pawar et al.[17], our analysis revealed a reduced proportion of CNS symptoms brought on by HIV-associated dementia and progressive multifocal leukoencephalopathy (PML). Neurotuberculosis involvement as a proportion was almost identical. In our analysis, there were fewer instances of fungal diseases. There were no cases of neurosyphilis reported. Our investigation revealed greater rates of neurotuberculosis and lower incidences of toxoplasmosis and fungal infections when compared to non-Indian studies by Sokolska et al.[18] and Berhe et al[19]. Nonetheless, the percentage of dementia linked to HIV is rather similar. In our analysis, CNS tuberculosis accounted for 33.33% of all cases, making it the most prevalent pathology overall. Ten percent of patients had tubercular abscesses, and forty-five percent had increasing parenchymal lesions and tuberculomas. 40% of cases had hydrocephalus that could be communicated. 6.67% of the cases had visible vascular infarcts, and 1.67% also had further evidence of arachnoiditis. Our study's findings about the spectrum of CNS TB in HIV patients are consistent with several other studies, including those by Sarosh et al.[20] and Michelle Whiteman et al.[21] Our study's imaging results for instances with fungal infections, CNS lymphoma, progressive multifocal leukoencephalopathy, Miguel J. et al., Enting et al., Mark et al., and HIV encephalopathy were consistent with earlier research by these authors.[22,23] Meningitis was present in 18.33% of the 60 patients. CNS tuberculosis was linked to every episode of meningitis. Aseptic meningitis and meningitis brought on by other opportunistic illnesses were not observed. In 6.67% of the patients in our investigation, an infarct-related imaging characteristic was present. In this specific cohort, HIV Vasculopathy was thought to be the diagnosis when other potential risk factors for CV stroke were ruled out and no clear etiological factors were found. It was not ruled out, though, that infarcts could have resulted from other infections, such as fungal infections, varicella-zoster virus, CMV, or a prior subclinical CNS infection. The cause of vasculopathy in HIV-positive people has been extensively discussed and is most likely multifaceted. Practically speaking, HIV-related vasculitis can be used to categorise HIV vasculopathy. MR angiography revealed a minor abnormality in the ipsilateral MCA in a single incidence of subacute ischemia. After a more thorough history was taken, the patient revealed a history of hypertension and heavy smoking.

CONCLUSION

In India, tuberculosis remains the most frequent CNS pathology associated with HIV/AIDS, despite an increasing tendency in HIV-associated primary neurological disease cases. While the imaging features of several brain disorders may overlap, specific imaging traits and the location of the lesions may support a specific diagnosis. However, in addition to imaging, linkage with clinical history and other laboratory markers is important. When a patient has neurological complaints and is seropositive for Acquired Immunodeficiency Syndrome (AIDS), magnetic resonance imaging (MRI) is a great way to identify and characterise brain lesions. If there are no contraindications, MRI should be one of the initial lines of study.

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