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Original Research Article

A CLINICAL STUDY ON NEUROLOGICAL MANIFESTATIONS IN HIV POSITIVE PATIENTS ATTENDING TO A TERTIARY CARE HOSPITAL

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ABSTRACT

Background: HIV is primarily spread through intravenous drug use while sharing needles and syringes, through heterosexual and intersex sexual contact, through blood and blood products, through breast milk, and occupational exposure to laboratory and healthcare workers. **Objectives:** 1. To study the spectrum of neurological manifestations in HIV-infected subjects at various levels. 2. To study the correlation of neurological manifestations with CD4 count in these subjects.
Material and Methods: Hospital-based prospective cross-sectional study. **Study area:** Department of General Medicine, **Study Period:** 1 year. **Study population:** Patients admitted to the General Medicine Nimra Institute of Medical Sciences wards, Vijayawada, Andhra Pradesh. **Sample size**: The study consisted of a total of 100 subjects. **Sampling Technique:** Simple Random sampling. **Study tools and Data collection procedure:** HIV patients admitted in a tertiary medical college were chosen for the study. A random selection of patients was made in whom a detailed history and clinical evaluation, including the mini-mental score (MMSE), was done after informed consent from the patient or relative.
Results: The mean CD4 Count of the patients who expired was 108.38. The mean CD4 count of the patients who improved were 231.78 and 268.60. There is a statistically significant correlation of CD4 count among patients who expired (P-0.003**). There were 10 Patients diagnosed with TB Meningitis. CD4 Count for one Patient was not done as the patient died before blood was taken for CD4 count. The mean CD4 count of patients with TB Meningitis in the study group was 120.88, and the mean CD4 count of patients without neurological manifestations was 225.02. Statistically significant differences in CD4 count were observed between the two groups. (P-0.025*).
Conclusion: The incidence of neurological illness in HIV infection in our study was 31%. All patients in our study had a heterosexual transmission of the disease. CNS manifestations in men were more common than in women. Headache and altered mentation were the two common symptoms observed in this study. Tuberculous meningitis was the most common opportunistic infection in our study. No significant CD4 count correlation was found between the patients with neurological manifestations and those without neurological manifestations.
Key Words: AIDS, CD4 count, Cryptococcal meningitis, HIV, Tubercular meningitis.

INTRODUCTION

A worldwide pandemic, HIV/AIDS (human immunodeficiency virus/acquired immunodeficiency syndrome), exists. According to the UNAIDS assessment of the worldwide AIDS epidemic in 2020, 38.0 (31.6-44.5) million persons were predicted to be HIV-positive in 2019.[1] Since the epidemic's peak in 2000, when the estimated adult HIV prevalence (15-49 years) was estimated at 0.55%, it has decreased to 0.32% in 2010 and 0.21% in 2021 on a national basis. The Southern States (0.67% in Andhra Pradesh, 0.47% in Telangana, and 0.46% in Karnataka) and northeast area States (2.70% in Mizoram, 1.36% in Nagaland, and 1.05% in Manipur) have the lowest adult HIV prevalence rates. About 24 lakh people are thought to be HIV-positive (PLHIV) at any given time. The top three states with the highest percentage of PLHIV are Maharashtra, Andhra Pradesh, and Karnataka.[2]

HIV is primarily spread through intravenous drug use while sharing needles and syringes, through heterosexual and intersex sexual contact, through blood and blood products, through breast milk, and occupational exposure to laboratory and healthcare workers. The primary cause of the growing quantitative and qualitative deficit of the subset of T lymphocytes known as helper T cells that characterize HIV illness is severe immunodeficiency. The CD4 molecule, which acts as the main cellular HIV receptor, is present on the surface of the helper T cell, which gives it its distinctive phenotypic characteristics.[3]

HIV-related immune deficiencies lead to infectious consequences. The primary mechanism is the increasing loss of CD4 T lymphocytes, which are essential for the immune system's general health. They multiply and release cytokines in response to antigen presentation. The interleukin (IL)-2 and interferon (IFN) gamma are two of the most significant. IFN gamma boosts antibody formation in B cells as well as the cytotoxic effects of natural killer cells and macrophages to act against intracellular pathogens, whereas IL-2 stimulates cytotoxic T cells that destroy viral infections. HIV-infected people are particularly at risk for both primary and reactivated TB due to impaired macrophage function brought on by decreased IFN gamma production.[4-9]

A measure for the probability of opportunistic infection is the CD4 lymphocyte count. The risk is minimal for people whose CD4 counts remain continuously above 200 cells/ml, but it rises when the CD4 count drops. While the immune system is functioning, many of the organisms that can cause infections are kept in check; however, as immunosuppression worsens, these organisms start to reappear. Examples include cytomegalovirus (CMV), EBV, and herpes zoster. Others, like Pneumocystis jiroveci pneumonia, candidiasis, recurrent bacterial infection, wasting syndrome, cytomegalovirus, lymphocytic interstitial pneumonitis, tuberculosis, nontuberculous mycobacteriosis, and herpes simplex virus, can develop de novo if the immune system is sufficiently weakened.[10-11]

As already mentioned, the CD4 count is used to assess how HIV is developing. All individuals who are initially diagnosed with HIV illness should have CD4 counts performed. Every three to six months, all HIV-positive patients are tested. Results can shed light on a potential AIDS diagnosis and the likelihood of opportunistic infections. The test also shows whether a treatment has failed. Before CD4 counts fall below 200 cells/mm3, antiretroviral medication (ART) should be started since problems are more common in this patient population. After beginning ART, levels should be checked every three to six months to see how the treatment is working. Every six to twelve months, the CD4 count can be rechecked if the response is appropriate.[12,13]

Hence the present study was undertaken to study the spectrum of neurological manifestations in HIV-infected subjects at various levels in the study population.

Objectives

1. To study the spectrum of neurological manifestations in HIV-infected subjects at various levels.
2. To study the correlation of neurological manifestations with CD4 count in these subjects.

MATERIALS AND METHODS

Study Design: Hospital-based prospective cross-sectional study.

Study area: Department of General Medicine, Nimra Institute of Medical Sciences, Vijayawada, Andhra Pradesh.

Study Period: 1 year.

Study Population: Patients admitted to the wards of the General Medicine and Neurology wards were subjects of the study.

Sample Size: The study consisted of a total of 100 subjects.

Sampling Technique: Simple Random sampling.

Study tools and Data collection procedure: HIV patients admitted in a tertiary Medical college were chosen for the study. A random selection of patients was made in whom a detailed history and clinical evaluation, including the mini-mental score (MMSE), was done after informed consent from the patient or relative.

The following investigation was done on all patients studied (i.e.) when tested positive for HIV.

1. Complete blood count.
2. Renal function test (Sugar, Urea, Creatinine and Electrolytes)
3. Liver Function Test (Bilirubin, AST, ALT, SAP, Albumin)
4. Chest X-ray - P.A View
5. VCTC
6. CD4 count
7. VDRL

All patients with neurological systems involved were subjected to the investigation listed based on clinical findings.

1. C.S.F.
2. CT Brain
3. MRI Brain
4. Creatinine Phosphokinase

Methodology of Investigation: HIV testing and CD4 count were done by the microbiology department in our hospital as per NACO Guidelines. CD4 count was done with Facs Count (Automated Counter). Tests were done in a single laboratory by the same person, and no interpersonal error was possible.

Exclusion Criteria: Immuno-compromised state due to any other cause.

Statistical Analysis: The Excel and SPSS (SPSS Inc, Chicago) software packages were used for data entry and analysis. The results were averaged (mean + standard deviation) for continuous variable parameters and the number and percentage for discrete variables are presented in Tables. The students' test was used to determine whether there was a statistical difference. A "p" value of less than 0.05 was accepted as indicating statistical significance.

RESULTS

Incidence: 31 Patients had neurological manifestations among the 100 Patients studied. The incidence of neurological manifestations in HIV infection, in this study is 31%.

Of the 100 Patients studied 80 were male and 20 were Female. Among the 80 males, 28 had neurological manifestations and of the 20 Females, 3 had neurological Symptoms. [Table 1]

The majority of the patients in our study were between 31-40 years of age. Of the 60 Patients, who were in the age group of 31-40, 19 (31.6%) had neurological symptoms.

All Patients in this study group had Hetero Sexual behaviour as the mode of transmission.

Among 31 Patients who had neurological symptoms, 4 Patients had pulmonary TB as co-infection. 19 Patients in the study group who had neurological manifestations were Ambulatory (i.e. able to do their daily activities but not working), 9 were working and 3 were bedridden. [Table 2]

Figure 1: Headache

Figure 2: Altered Sensorium

11 Patients who had neurological manifestations in the study group expired (35.48%) and 20 Patients had improvement in their clinical condition. [Table 5]

CD4 count levels in Patients with neurological manifestations ranged from 11 and 535 with an average of 179.19. The average CD4 levels in Patients without neurological manifestations was 225.03. There was no statistically significant difference between the two groups. (P>0.05). [Table 6]

The mean CD4 Count of the patients who expired was 108.38. The mean CD4 count of the patients who improved were 231.78 and 268.60. There is a statistically significant correlation of CD4 count among patients who expired (P-0.003**). [Table 7]

There were 10 Patients diagnosed with TB Meningitis. CD4 Count for one Patient was not done as the patient died before blood was taken for CD4 count. The mean CD4 count of patients with TB Meningitis in the study group was 120.88 and the mean CD4 count of patients who did not have any neurological manifestations was 225.02. A statistically significant difference in CD4 count was observed between the two groups. (P-0.025*). [Table 8]

Mini-mental Score of Patients with neurological symptoms was compared with those without neurological symptoms. A significant difference was observed in patients with neurological symptoms, the P value being less than 0.001.

Fundus: Fundus Examination was done for all co-operative patients. 10 patients had features of papilledema and no patient had features of HIV Retinitis.

CSF Analysis:

Analysis was done for 20 patients in the study.

11 Patients had elevated proteins and predominant lymphocytes. 4 Patients had normal CSF.

2 had elevated proteins and acellular smear. 2 patients had predominant neutrophils.

1 had elevated proteins and occasional lymphocytes and also had positivity for cryptococcus in India Ink preparation.

CT Brain: 24 Patients in the study were subjected to CT Brain among which 3 patients had middle cerebral arterial territory infarct and 1 had multiple calcified granulomata.

MRI: In this study 4 patients had MRIs brain done for them. 1 had multiple ring-enhancing lesions and was diagnosed with Tuberculoma and other patients had features suggestive of normal pressure hydrocephalus.

Table 1: Shows the sex distribution in this study

Sex	Positive	Negative	Total
Male	28	52	8 0
	35%	65%	80%
Female	3	17	20

Table 2: Shows the age distribution in this study

Age in Years	Positive	Negative	Total
<30	4 25%	15 75%	19
31-40	19 31.6%	41 68.3%	60
>40	8 38.09%	13 61.91%	21

Table 3: Clinical presentation

Clinical Presentation	Frequency	Percentage
Headache	22	38.6%
Altered Sensorium	14	24.56%
Hemiplegia	3	5.26%
Seizures	8	14.03%
Paraparesis	4	7.01%
Quradriperesis	2	3.5%
Parasesthesias	2	3.5%
Cerebellar Syndrome	2	3.5%

Table 4: Shows the disease patterns among patients who had neurological manifestations

Diagnosis	Frequency	Percentage
Acute Flaccid Paralysis	1	3.22%
AIDS Dementia Complex	1	3.22%
Cerebellar Syndrome	1	3.22%
Cryptococcal Meningitis	1	3.22%
Toxoplasmosis	1	3.22%
Cerebrovascular Accident	3	9.67%
Guillian Barre Syndrome	1	3.22%
HIV Myelopathy	1	3.22%
Meningoencephalitis (Cause not determined)	1	3.22%
Multiple Granuloma	1	3.22%
Myopathy	1	3.22%
Peripheral Neuropathy	2	6.44%
Pyogenic Meningitis	2	6.44%
Seizure Disorder	2	6.44%
TB Meningitis	11	35.4%
Tuberculoma	1	3.22%

Table 5: Shows the outcome of the study

Out Come	Frequency	Percentage
Improved	20	64.52%
Expired	11	35.48%

Table 6: CD4 count

	CD4 Count	
	Mean	SD
Patient with Neurological Manifestation	179.19	113.16
Patient without neurological Manifestation	225.03	134.58

Table 7: CD4 Relation with Mortality

Out Come	CD4 Mean	Count SD
Expired	108.38	41.65
Improved	268.60	110.94

Table 8: CD4 Relation with TB Meningitis

	No. of Cases	Mean	SD
Patients with TB Meningitis	11	140.88	43.161
Patients without Neurological Manifestation	69	245.02	138.585

Table 9: Mini Mental Score

	MMS	
	Mean	SD
Patients with Neurological Manifestation	24.94	2.13
Patients without neurological Symptoms	26.65	.84

DISCUSSION

In this study, of the 100 seropositive patients, 31 had neurological manifestations (31%). When compared with the findings of Wadia et. Al,[14] was on par who reported an incidence of 24.8% in his report but was on low with the findings of Millogo et,[15] who reported a 15% incidence in his study. Most of the patients in this study were in the age group of 31 - 40
(59.38%). The mean age of the patients with neurological manifestations in a study in the University of California and San Fransisco data was 37.3 years. The mean age in this study was 35.63 years. Male patients were found to have neurological manifestations more common (90.63%) than females (9.38%). Male to female ratio was approximately 9:1. Metha et al,[16] have reported male predominance with male to female ratio of 12:1.

All patients with neurological manifestations had heterosexual behaviour as the risk factor. None of our patients had a homosexual relationship. Gupta et al,[17] found heterosexual relationships in 64.7%, 5.85% of drug abusers and blood transfusion in 14.7%.

This was the commonest presenting symptom in this study. 22 of the 31 patients with neurological symptoms, presented with headache (38.6%).[16] patients (72.72%) had opportunistic infections like tuberculous meningitis, cryptococcosis and pyogenic meningitis as the cause. Six other patients had HIV dementia and multiple granulomata as the cause. Headache is a ubiquitous symptom in HIV infection, because of the frequency of intracranial infection and mass lesions. Saag, Gray Bill et al,[18] have described headaches as a common symptom of HIV infection frequently.

14 out of the 31 patients, had altered sensorium (24.6%). Altered sensorium as observed in this study was primarily due to a meningeal infection, tuberculous meningitis being most frequent, followed by cryptococcal and pyogenic meningitis. None of the patients in this study had CNS lymphoma. The University of California and San Fransisco data revealed altered sensorium as a manifestation of secondary viral infection. Progressive multifocal leukoencephalopathy, toxoplasmosis, cryptococcosis, HIV dementia and lymphoma.

In our study, the common cause of seizures was neurotuberculosis. Of the 31 patients, 8 patients had seizures (14%). Two patients had normal CT brain and C.S.F analysis did not reveal any abnormality. EEG could not be tested on the above patients. This is perhaps because approximately half the HIV-infected patients have no definite identifiable disease of the brain and cerebral HIV infection seems to be the likely cause of the seizures, as reported by Holtzman et al,[19] AMJ Med. study which had HIV encephalopathy as the cause of seizures in 24% of the patients.

4 patients had paraparesis on presentation in our study (7%). Of the 4, one was due to Guillian-Barre syndrome and 3 due to HIV myelopathy. Human T cell lymphotropic virus 1, tuberculosis, herpes zoster and syphilis were the causes of paraparesis described by A. I Bhigjee et al,[20] in their study. Two patients in our study had paresthesias of both lower limbs (3.5%). One patient was on antiretroviral therapy (which included zidovudine) and another patient was not on ART. Both were improved with Amitryptiline and nutritional support. Fuller, Le Fauchur et al,[21] has shown an incidence of 9.46% of peripheral neuropathy in his study.

Of the 31 patients, 1 had features of cerebellar syndrome (3.22%). One patient had a hypodense lesion in the cerebellar area in the CT brain (plain). Among the 31 patients, one had AIDS dementia complex (3.22%). The patient presented with a headache and progressive cognitive decline. The minimal score of the patient was 20. CT brain and C.S.F analysis were normal. Impaired memory and concentration with psychomotor slowing represent the common early presentation of this disorder. Vijay D. Teja et al,[21] reported an 8.03% incidence in their studies. Given these findings baseline MMSE is probably advisable for all cases with HIV seropositivity and periodic evaluation may unearth more cases with AIDS dementia complex.

In our study, two patients had peripheral neuropathy (6.44%). Both the patients presented with paresthesias and were foot drop. One was taking antiretroviral therapy which included zidovudine and the other was not on antiretroviral. HIV-associated sensory neuropathies include both distal sensory polyneuropathy due to HIV infection and antiretroviral toxicity. It is very difficult to differentiate between the two. Treatment is largely symptomatic. Our patients improved with a change of retroviral regimen, Amitryptiline and nutritional support.

One patient in our study had myopathy (3.22%). The patient presented with myalgia and proximal muscle weakness. On investigating further, he had elevated creatinine kinase levels. The patient was on antiretroviral therapy. After stopping zidovudine patient was followed up, with improvement in symptoms. Studies have suggested that zidovudine-induced myopathy occurs only when an underlying HIV-related inflammatory myopathy is present.

Of the 31 patients in the study, two had HIV-related myelopathy (6.44%). The patient had lower limb weakness and urinary incontinence on presentation. MRI spine showed no abnormalities and C.S.F analysis was normal. Jerez.p et al,[23] have shown a 22% incidence of spinal lesions in AIDS. The leading cause of myelopathies described in association with HIV was vacuolar myelopathy followed by myelitis.

12 out of 31 patients who had neurological symptoms in our study had tuberculous infection of the nervous system. Among the 12 patients, 11 had tuberculous meningitis (35.4%) and 1 had tuberculoma (3.22%). 3 of the 11 patients with tuberculous meningitis expired (27.27%). The patient who was diagnosed with tuberculoma had presented with headaches and partial seizures involving the left upper limb. MRI brain revealed multiple ring-enhancing lesions.

C.S.F analysis showed elevated protein with predominant lymphocytes. The increased number of Neurotuberculosis in Indian studies is probably due to the high prevalence of tuberculosis in this part of the world. In all our patients tuberculous meningitis was the first manifestation of the disease. Among them five patients on Antiretroviral therapy.

2 patients had features of pyogenic meningitis (6.44%). Both the patients presented with altered sensorium, fever and headache. CT brain was normal and C.S.F analysis revealed elevated proteins and predominant neutrophils in cytology. C.S.F culture did not grow any organism.

Both the patients however succumbed to the disease proving the point that pyogenic infection coexisting with HIV infection has a very high mortality. Of the 31 patients, one had cryptococcal meningitis (3.22%). Headache, altered sensorium and signs of meningeal irritation were the presentations. C.S.F analysis in this patient was positive for Cryptococcus on India ink preparation. Of 31 patients one had toxoplasmosis (3.22%). Headache and fever were the presentations. IgG Ab positive for Toxoplasmosis and MRI was normal. 3 patients in our study presented with cerebrovascular complications (9.66%). All three patients presented with hemiparesis and their CT brain showed middle cerebral arterial territory infarct. On admission, he expired on the same day. Evaluation of the 3 patients for young stroke showed no abnormalities. Gupta et al,[17] reported an 8.82% incidence of CVA in seropositive patients in their study.

Among the 31 patients with neurological symptoms, 1 had Guillain–Barre syndrome (3.22%). Both the patients presented with features of ascending paralysis. C.S.F analysis showed elevated protein with acellular smear cytology. A nerve conduction study and Oligoclonal band in C.S.F could not be done. Two patients in this study group presented with seizure disorder (6.44%). One patient presented with a generalized seizure. CT brain and C.S.F analysis were normal in both patients. EEG could not be done. In our study one patient presented with a chronic headache of 6 months duration. CT brain done on the patient showed multiple calcified granulomas. C.S.F analysis revealed no abnormality. As the patient could not afford MRI brain was not done. The patient was empirically treated with albendazole with no apparent improvement on immediate follow-up.

20 out of the 31 patients who had improved outcome (35.48%). As improved Antiretroviral Therapy, nutritional and social support continues to the prolong life span of HIV-infected persons. In line with our investigation, studies have also noted the occurrence of neurological

complications and other clinical symptoms linked to a low CD4 count and a high viral load. Secondary symptoms were more frequent in those with CD4 levels under 100 cells per millilitre. The mean CD4 count of the study group's cases was 218.085.3cells/mm3, which is nearly identical to the mean count reported by Mansuri ZH et al,[24] who reported it to be 228.038.14cells/mm3. However, the prognosis was poor in instances with CD4 levels below 100 cells/mm3. Regardless of CD4 numbers, tubercular meningitis was the most prevalent symptom in our sample.

CONCLUSION

The incidence of neurological illness in HIV infection in our study was 31%. All patients in our study had a heterosexual transmission of the disease. CNS manifestations in men were more common than in women. Headache and altered mentation were the two common symptoms observed in this

study. Tuberculous meningitis was the most common opportunistic infection in our study. No significant CD4 count correlation was found between the patients with neurological manifestations and those without neurological manifestations. CD4 count when less was associated with increased mortality. Patients with coexisting tuberculous meningitis and HIV infection had significantly lower CD4 counts. Tuberculous meningitis was associated with good outcomes and pyogenic meningitis had high mortality. Patients with neurological manifestations had good outcomes and low mortality.

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