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Original Research Article

Incidence of brain parenchymal abnormality in a group of HIV positive patients admitted in tertiary care hospital: a prospective descriptive study

S. K. Nath[1]*, S. M. Sudumbrekar[2], Sumeet Arora[3], S. K. Rai[4]

[1]Department of Radiology, Military Hospital Devlali, Nashik, Maharashtra, India
[2]Department of Neurosurgery, Command Hospital, Chandigarh, India
[3]Department of Medicine, Army College of Medical Sciences, New Delhi, India
[4]Department of Orthopaedics, 151 Base Hospital, Guwahati, Assam, India

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***Correspondence:**
Dr. S. K. Nath,
E-mail: saumennath73@gmail.com

ABSTRACT

Background: Magnetic resonance imaging (MRI) has been used to examine the impact of human immunodeficiency virus (HIV) on the central nervous system (CNS) since the beginning of the disease. The objectives of this study were to determine the incidence of brain parenchymal abnormality in a group of HIV positive patients and to detect and study the profile of MRI patterns of brain lesions in HIV positive patients.
Methods: In our study, total of 28 patients have been evaluated enrolled between 01 December 2013 to 30 December 2014.
Results: During the study period 35 patients on immune-surveillance were screened for brain lesions of which 28 patients met the inclusion criteria and were included in study. The male-female ratio has been found to be 4.6:1 with the mean age of 43 (18-77). Majority of patients presented with headache as main clinical symptom. Out of which 20% normal, 13.3% NSWM, 13.3% atrophy, hematoma 6.6%, infarct 6.6%, toxoplasmosis 13.3%, PML 13.3%, NCC 6.6% and CMV encephalitis 6.6%. Altered mental status and neurological deficit 27.6% and 20.7%, respectively, were two other symptoms following headache. MR imaging detected neuroparenchymal abnormality in 20 (71.4%), out of 28 HIV positive patients. There was no neuroparenchymal abnormality seen in the rest of the 08 patients
Conclusions: MR imaging detected neuroparenchymal abnormality in 20 (71.4%), out of 28 HIV positive patients. Based on our study we can conclude that the MRI of brain is the primary modality to detect the brain lesion in HIV positive patient even if he is asymptomatic clinically.

Keywords: Brain parenchymal lesion, HIV infection, MRI brain

INTRODUCTION

Magnetic resonance imaging (MRI) has been used to examine the impact of human immunodeficiency virus (HIV) on the central nervous system (CNS) since the beginning of the disease. Consequently, our understanding of the evolution and progression of CNS injury in the context of HIV has grown tremendously. However, the specific role of imaging over the years has yet to be realized in the current era of treatment where many questions remain regarding the evolution and progression of HIV-associated CNS injury. White matter abnormalities are frequently seen on brain MRI of HIV positive (HIV+) patients. We aimed to determine the

prevalence of unexplained White matter abnormalities and their associations with HIV disease.

Imaging analysis and diagnostic criteria (DC) of Brain lesions

Acquired Toxoplasma encephalitis

This is characterised by fever, malaise, headache, personality changes and seizures at later stages. DC- Ill-defined hypointense lesions (occasionally hyperintense) on T1W1. Hypointense and hyperintense peripheral edema on T2W.[1] Target sign in FLAIR. Increased diffusivity in necrotic centre on DWI. Rim/ Nodular/ Punctate enhancement. Prominent lipid peak on MRS.

Acquired CMV Encephalitis

May present as Meningitis, Encephalitis, Ventriculitis, Transverse myelitis, Radiculomyelitis and chorioretinities. DC: on T1W1 Encephalitis: Hypointense mass, Ventriculitis: enlarged ventricles with debris level. On T2W1 and FLAIR, Encephalitis: hypointense periventricular mass, Ventriculitis: enlarged ventricles with surrounding hyper intensity. Enhancement exhibited only with necrosis/ encephalitis. Ependymal and periventricular enhancement inVentriculitis. Necrotising encephalitis may show increase in choline, Increase in lactate peak in MRS.[2]

HIV encephalitis DC

This is characterised by focal abnormalities of increased T2 signal intensity or diffuse moderate high signal intensity WM changes. FLAIR can pick <2 cm lesions. Cerebral atrophy. No enhancement on PCI. Decrease NAA and increase choline peak on MRS.[3]

Cryptococcosis

It usually spread along perivascular spaces in CNS-haematogenous dissemination from lungs. Headache most common symptom. DC: Perivascular spaces are filled with fungi, isointense to CSF on T2W1. FLAIR may exhibit small hyperintense rim in these lesions. These cysts also may form gelateneous pseudocysts (hyperintense) in basal ganglia, thalamus, brainstem, cerebellum, periventricular and subcortical WM. Rarely military or leptomeningeal enhancing nodules seen.[4]

Progressive multifocal leukoencephalopathy

This is seen when JC virus infects oligodendrocytes, causes demyelination in immunocompromised patients. DC: Aggressive and burnt out PML lesions appear hypointense on T1W1. On FLAIR and T2W1, hyperintense lesions predominantly involve the subcortical U-fibres and periventricular WM. Only new lesions exhibit slightly restricted diffusion. Faint peripheral enhancement rarely seen in patients with long time survival and patchy enhancement may be seen in IRIS (PML-IRIS). On MRS, decreased NAA peak and increased lactate, choline and lipid peaks.[5]

Immune reconstitution inflammatory syndrome

This is characterised by Atypical / worsening opportunistic infection in HIV/AIDS patients following commencement of HAART (1/4th cases), JC virus and Mycobacterium Tuberculus being common. DC: Hypointense T1W1 and hyperintense on T2W1 lesions increase in size, enlarge and become confluent, exert mass effect (PML IRIS) and increase edema around the tuberculomas (TB IRIS) seen. Patchy atypical enhancement (PML IRIS), increase in size of ring / nodular enhancing tuberculomas with increase pial enhancement (TB IRIS) and increase nodular meningeal/ subependymal enhancement with increase in size of gelatinous pseudocysts (Crypto-IRIS).[6]

Primary CNS lymphoma (PCNSL): diagnostic criteria

Isointense to hypointense to cortex on T1 and T2W1 with mild surrounding edema. May appear heterogeneous on T2W1 due to haemorrhage /necrosis. Variable restricted diffusion and peripheral enhancement if central necrosis. Decrease NAA, increase choline on MRS.[7-9]

Ischemic stroke/infarct

In HIV patients, the coagulation necrosis is due to vasculopathy and mass affect.[10] DC: FLAIR - most sensitive. Restricted diffusion may be diagnostic. Blood degradation products in GRE sequences. Relative age of a cerebral infarct can be determined with post contrast studies - intravascular contrast enhancements (immediate) Meningeal enhancement adjacent to infarct (12-24 hours), Early parenchymal contrast enhancement seem (1-3 days), Intravascular and meningeal enhancement begin decreasing (4-7 days) and intravascular and meningeal enhancement disappear with striking parenchymal contranst enhancement.[11-13]

Neurocysticercosis

Immunocompromised patients are prone to Taenia Solium. Clinically it may present with headache, seizure or hydrocephalous. DC as per 4 developmental stages.[14] On T1WI, T2WI and FLAIR: Vesicular-Isointense to CSF cystic lesion with ± discrete eccentric T1hyperintense scolex. Colloid vesicular - Cyst mildly hyperintense to CSF. Granular nodular - Cyst wall is thickened and retracted. Edema decreases. Nodular calcified- Shrunken lesion. GRE sequences are useful to pick calcified scolex. Enhancement is seen - scolex in Vesicular, thick cyst wall and marginal nodule in Colloid vesicular and nodular or ring like enhancement of the thickened retracted cyst wall in Granular nodular. Few cases exhibit increased lactate, alanine, succinate, choline; decreased NAA and creatinine on MRS.

METHODS

Study design

All HIV positive patients on immunosurveillance admitted in Base Hospital, New Delhi, India, were included during 1yr study period from 01st December 2013 to 30th December 2014. Total of 28 patients have been evaluated, enrolled in this prospective descriptive study, conducted at tertiary care hospital.

Inclusion criteria

- Patient of either sex or all age group
- Symptomatic patients presenting with headache, vomiting, seizure, altered mental status, neurological deficit, aphasia, altered mental status, dementia, meningism and visual impairment
- Asymptomatic patients are those with CD4 count <200/ µL.

Exclusion criteria

Patients with cardiac pacemaker, metallic FB in eye, implants etc.

Table 1: MRI protocol.

Protocol of Sequences	Sag	FRFSET2
	Axial	T2 Propeller, T1FLAIR, T2 FLAIR
	Cor	FRFSET2
	DWI and T2 GRE images in axial planes	
Additional sequences	Post Gad T1 SE in three planes	
	MVMRS	
	MRA	
	MRV	

Methodology

Clinical Assessment

Patients admitted with brain lesions were followed up from the beginning to study clinical pattern and to detect early deterioration. Age, gender, co-morbidity, coexistence of other major illness, alcohol consumption, history of ART, blood transfusion and low CD4 count were enquired into. Data collected from all patients meeting the inclusion criteria.

Imaging

MRI was conducted on GE HDX 1.5 Tesla machine. Image analysis was done using the image processor AW MR Advantage Windows 4.4 volume share, which has multiplanar reconstruction capability. Protocol of MRI sequences as per the standard parameter followed globally as shown in Table 1.

RESULTS

Clinical presentation

Majority of patients presented with headache as main clinical symptom as in Figure 1. Altered mental status and neurological deficit 27.6% and 20.7%, respectively, were two other symptoms following headache.

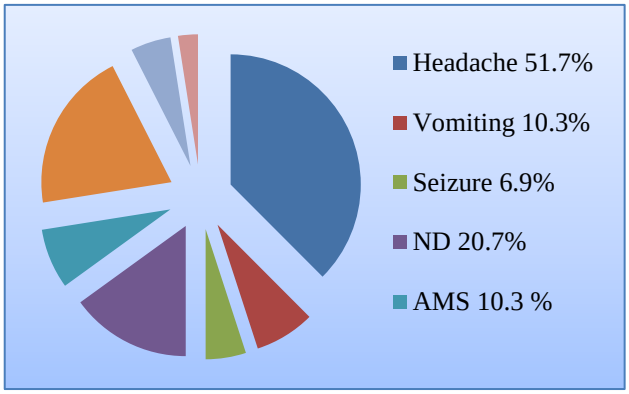

Figure 1: Clinical presentation and sex distribution.

Discrete and confluent lesions appearing hypointense/isointense in T2 and T1WI with some lesions appearing hyperintense on T2WI with perilesional edema seen in right lentiform nucleus, left centrum semiovale and at GW junction in bilateral frontal and right insular regions. PCI = ring/nodular enhancement. On MVMRS, lipid-lactate peak is noted with choline/creatine ratio= 0.9; and NAA/creatine ratio = 1.6.

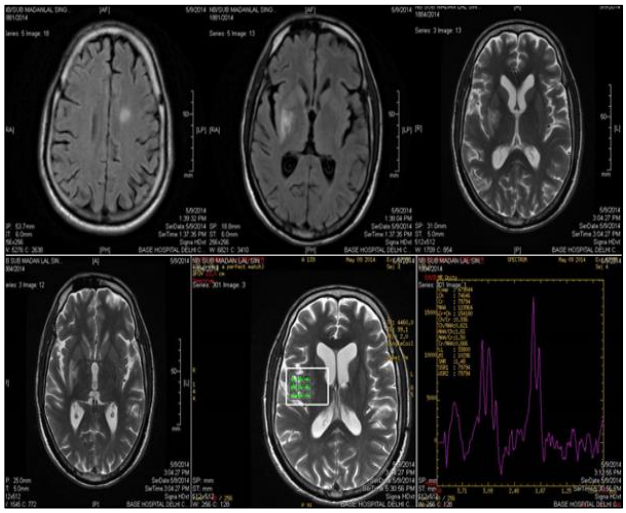

Figure 2: MRI picture of Toxoplasmosis.

Table 2: Demographical details, clinical presentation, and MRI finding.

Pt.	Age	Sex	Clinical presentation	MRI findings							Impression
				T1 W1	T2 WI	FLAIR	DWI ADC	GRE	PCE	MRS	
1	53	M	Lt hemiparesis, headache	-	+	+	RD	B	0	NA	Infarct Rt MCA territory.
2	37	M	Hemiparesis	-	+	+			p	Ccr 1.45 Ncr 0.8	PML
3	48	M	Headache	0	0	0	0	0	0	NA	Cerebellar and cerebral atrophy
4.	32	M	Lt Hemiparesis	-	+	+	0	0	0	NA	Chronic infarct Rt MCA territory
5.	50	M	Headache	MSI	MSI	MSI	0	0	0	Ccr 0.9 Ncr 1.6	Toxoplasmosis
6.	65	M	Headache, Vomiting	-	+	+	0	0	0	NA	PML
7.	35	F	Headache	Iso	MSI	MSI	RD	0	0	NA	NCC
8.	31	M	Headache	Iso	+	+	0	0	0	NA	PML
9.	42	M	AMS	Iso	+	+	0	0	0	NA	HIVE
10.	29	F	Headache	_	+	0	0	0	NA	NA	Normal study
11.	30	M	Headache	Iso	+	+	0	0	0	Ccr 1.92 Ncr 1.12	CMVE
12.	50	F	Solitary seizure	_	_	_	0	0	RE,NE	NA	NCC
13.	39	M	Seizure	MSI	+	+	0	B	PHE	P	Lymphoma
14.	59	M	-ve response, aphasia	_	+	+	RD	0	RE, PHE	P	PML- IRIS
15.	77	M	Giddiness, Atonia	_	+	+	0	0	0	NA	Chr.infarct left periventricular region
16.	50	M	Headache, vomiting	_	+	+	RD	0	RE, PHE	P	Toxoplasmosis
17.	39	M	Headache, vomiting	+	+	+	0	B	RE, PHE	NA	Intraparenchymal hematoma
18.	18	M	FU cryptococcal meningitis	0	0	0	0	0	0	NA	Normal Study
19.	48	M	Headache	0	0	0	0	0	NA	NA	NSWM
20.	36	M	CD4 count 14	0	0	0	0	0	NA	NA	Normal study
21.	38	M	CD4 count 11	0	0	0	0	0	NA	NA	Normal study
22.	38	M	Headache	0	0	0	0	0	NA	NA	Normal study
23.	41	M	Neurocognitive deficit.	00	00	00	00	00	0NA	NA	Normal study
24.	60	M	Declining CD4 count	0	0	0	0	0	NA	NA	Normal study
25.	52	M	Headache	_	+	+	0	B	0	NA	NSWM
26.	45	M	Headache	0	0	0	0	0	NA	NA	Atrophy (Cerebral and cerebellar)
27.	35	F	Lt hemiparesis	_	+	+	RD	0	0	NA	Infarct right MCA territory
28.	38	F	PUO, CD4 68	0	0	0	0	0	NA	NA	Normal study

Ill-defined areas of altered signal intensity, fairly symmetrical, seen involving bilateral deep fronto-parietal WM appearing hyperintense on T2WI, FLAIR and isointense on T1WI- extending into left lentiform nucleus, head of left caudate nucleus, right thalamus, midbrain (right>left) and posterior limbs of bilateral

internal capsules and the region around anterior commissure. There is sparing of subcortical U fibers. No significant mass effect seen. PCI = No parenchymal or meningeal enhancement.

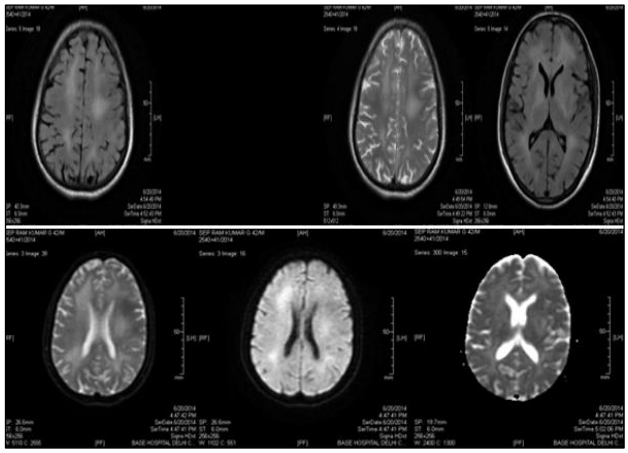

Figure 3: HIV Encephalopathy.

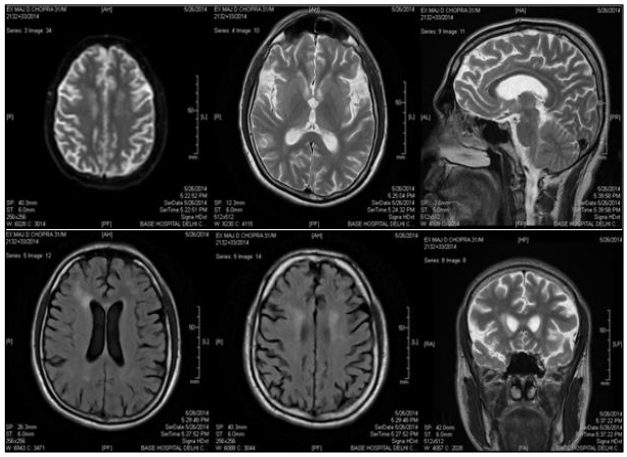

Figure 4: Diffuse cerebral and cerebellar atrophy: suggestive of PML.

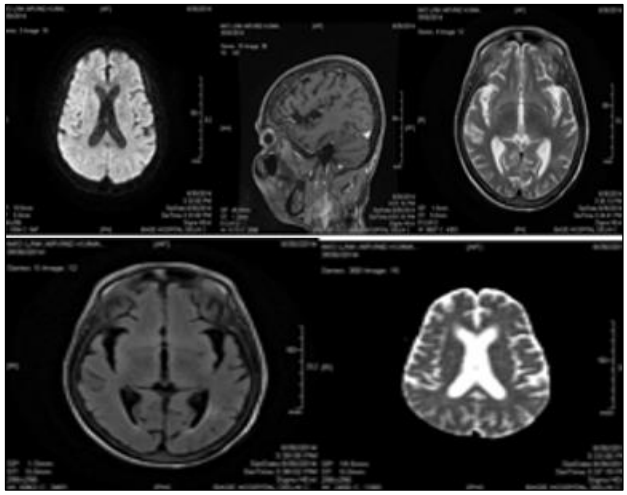

Figure 5: MRI picture of Neurocysticercosis at granular nodular stage.

A well-defined round lesion measuring 4 × 4.3 × 5.8 mm is noted at grey-white junction at left occipital region appearing hypointense with central isointense nodule on all pulse sequences. Ring enhancement on post contrast images. No perifocal edema seen. No blooming noted in SWI. No restriction of diffusion suggesting no acute lesion present.

Out of 28 cases studied, 24 were symptomatic. Amongst these 24, the MRI findings correlated with the clinical picture in 18 (75%) cases.

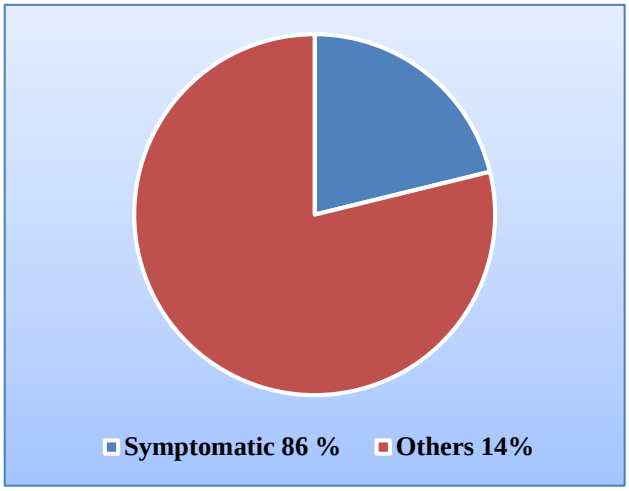

Figure 6: Percentage of symptomatic patients.

Detailed study of MRI images as per institutional protocol, 8 (28.6%) patients were found to have normal MRI findings as in Figure 7.

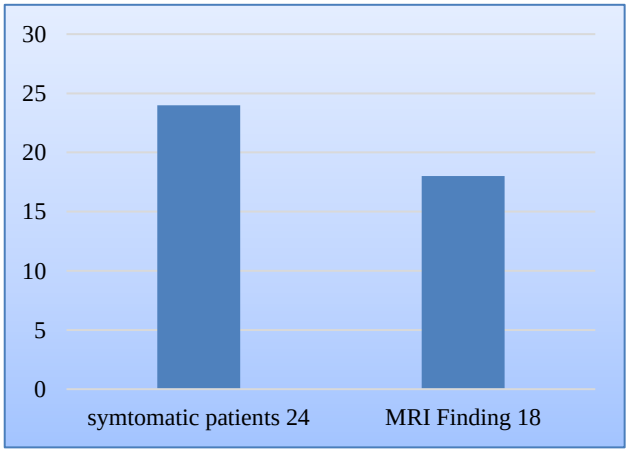

Figure 7: Percentage of MRI findings correlated with clinical pictures (75%).

Stroke detected in 5 (17.8%) patients in total, out of which 4 (14.3%) were infarct and 1 (4.3%) haemorrhagic stroke. Cerebral and cerebellar atrophy were detected in 2 (7.14%) patients whereas non-specific white matter changes are seen in 2 (7.14%) patients. Infective aetiology found in 10 (35.71%) cases, PML leading with 3 (10.7%) cases. Followed by 2 (7.14%) cases of NCC, 1

(3.5) case, each of CMV encephalitis and HIVE. Out of these, 1 (3.5%) case of IRIS has been detected, features likely suggesting PML-IRIS. Single case (3.5%) of malignancy, PCNSL has been detected during this study period as shown in Figure 8.

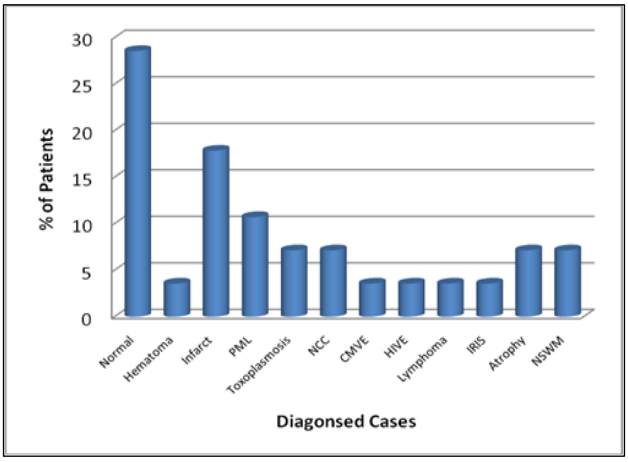

Figure 8: Frequency distribution for diagnosed cases.

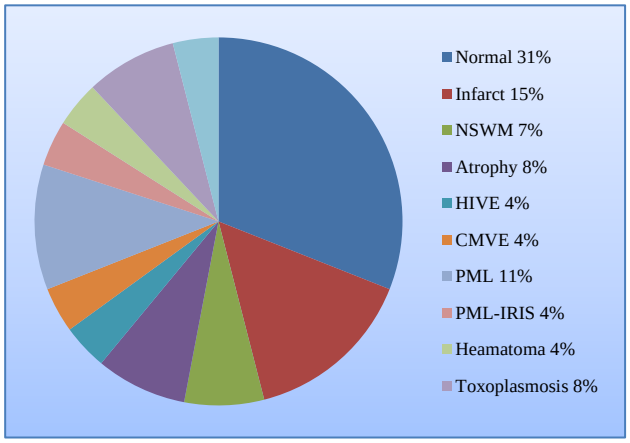

Figure 9: MR spectrum of neurological findings.

DISCUSSION

MRI is biomarker for brain lesions in HIV patients and plays important role in diagnosis and management of neurologic illness in AIDS.

However, normal studies does not exclude pathology e.g. initial cryptococcal infection. Appearance of new brain lesions in a previously normal imaging study with initiation of HAART is seen with PML, TB, and cryptococosis. In PML IRIS, which is more common amongst the above three, may exhibit appearance of post contrast enhancement.[15]

Brain atrophy

Brain atrophy was seen only in 2 (7.14%) cases in the present study Figure 9. However, study in a year 1992 find brain atrophy in 7% cases and in another study 8.7% with neuroparanchymal symptoms.[16,17]

NSWM

NSWM was found in 2(7.1%) cases in our study in comparison to 7(34.8%) a study of 19 patients.[18] In another study, 84% of 50 HIV positive patients were neurologically asymptomatic and 16% had mild cognitive impairment.[19]

PML

PML was found in 2 (7.1%) in the present study as shown in Figure 4. In another study which showed decrease in incidence with HAART.[20] In another study, 27% of the HIV positive patients developed PML. This study concluded a 12-fold increase in the frequency of PML between 1981-84 and 1991-94.[21]

PCNSL

This was found in 3.5% case in the present study. In a comparative study with CT and MRI, 82 lesions were identified with MRI findings in 22 patients.[22] In another study, 6% of HIV positive patients were diagnosed as having CNS lymphoma.[23] In another study, out of suspected PCNSL cases, 42.6% of patients, histologic diagnosis made by brain biopsy.[24]

Toxoplasmosis

This was found in 2 (7.1%) cases in the present study as shown in Figure 3. One of the studies with patients of CD4 counts below 50cells/µL, toxoplasmic encephalitis constitutes 19% of cases.[25] In another clinico-radiological study, 3 out of 49 patients had toxoplasmic encephalitis within a median interval of 369 days since the first diagnosis.[26]

HIV Encephalopathy

About 30% cases manifest disease characterized by progressive dementia followed later by pyramidal and cerebellar dysfunction.[27] Wherein, another study showed HIVE in 13 (5%) in evaluation of focal brain lesion.[28] In another study, 25 were detected to have HIV related encephalopathies in 60 HIV seropositive patients.[29] However the present study revealed single (3.6%) case as shown in Figure 5.

CMVE

It was found in one case (3.6%) in the present study without ruling out HIVE co-infection. Concomitant infection of CMV with HIV is seen in 6 (19.4%) pts and one had necrotizing ependimitis and meningoencephalitis.[30] In a study with 35 patients, 19 had diffuse WM lesions of which 3 (8.57%) had CMVE.[31]

Ischemic and haemorrhagic stroke

HIV associated vasculopathy was identified in 13 (20%) patients in one of the study.[32] In another study, the different mechanisms were worked out for different etiologies. Stroke mechanisms are variable in HIV-infected patients, with a relatively high incidence of vasculitis and hypercoagulability.[33] However only 4 cases (14.3%) diagnosed in the present study and one of the case was of RHD thus raising the possibility of embolic stroke. A single case (3.6%) of haemorrhagic stroke has been diagnosed during the study period.

Cryptococcosis

It was noticed that in 9 (36%) developed MRI +ve lesions in a study of microbiologically positive 25 patients.[34] In another study, out of 29 immunocompromised patients, 10 (34%) patients had been evaluated with abnormal MRI findings.[35]

Neurocysticercosis

It was difficult to diagnose due to deranged immunological parameters.[36] A case study with 18 patients showed 12% of NCC, all presented with epileptic attacks.[34] In the present study, we came across 2cases (7.14%) as in Figure 6.

Thus, in comparison to various studies conducted in different parts of the world, trends of various brain lesions in HIV positive patients are discussed. There is no significant increase in the incidence of brain atrophy over the years whereas incidence of non-specific white matter changes are definitely decreased. Use of newer MR parameters like MRS, diffusion tensor imaging, perfusion imaging etc, lesions are better characterised, and this may explain the decreasing trend in non-specific white matter changes. PML exhibits a decrease in incidence. Opportunistic infections appear to exhibit a decreasing trend except toxoplasmosis which shows a mildly increasing trend. Stroke, however, shows an increasing trend suggesting no significant effect of immunomodulation by ART. Lifestyle changes with co-morbities like atherosclerosis, hypertension, obesity, diabetes etc may contribute to the increased incidence in HIV positive patients. Out of 28 cases only one (3.6%) case of malignancy was diagnosed. Out of 28 cases, 24 were symptomatic. Amongst these 24, the MRI findings correlated with the clinical picture in 18 (75%) cases.

This is prospective study of small group of 28 HIV patients, however more study is needed and early detection by MRI is required in order to detect brain parenchymal changes in these patients.

CONCLUSION

MR imaging detected neuroparenchymal abnormality in 20 (71.4%), out of 28 HIV positive patients. There was no neuroparenchymal abnormality seen in the rest of the 08 patients. MRI thus has a good clinical utility in evaluation of neuroparenchyma in HIV positive patients.

The effects of HIV on the brain parenchyma, structural and functional, can be non-invasively assessed by MRI.

Positron emission tomography (PET) of glucose metabolism, neurotransmitter systems' abnormalities, or amyloid deposition could provide additional understanding of the neuropathophysiological changes associated with HIV.

Neuroimaging studies that are longitudinal; have larger sample sizes of both HIV infected (HIV+) and HIV uninfected (HIV-); and include HIV+ patients of different disease durations are needed. Novel neuroimaging methods could be added to current criteria for defining HIV associated parenchymal changes and neurocognitive disorders. These methods may also help in evaluating the efficacy of combination anti-retroviral therapy (cART) regimens.

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Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Masamed R, Meleis A, Lee EW, Hathout GM. Cerebral toxoplasmosis: case review and description of a new imaging sign. Clinical Radiol. 2009;64(5):560-3.
2. Miller RF, Lucas SB, Hall-Craggs MA, Brink NS, Scaravilli F, Chinn RJ, et al. Comparison of magnetic resonance imaging with neuropathological findings in the diagnosis of HIV and CMV associated CNS disease in AIDS. J Neurol Neurosurg Psych. 1997;62(4):346-51.
3. Letendre SL, Ellis RJ, Everall I, Ances B, Bharti A, McCutchan JA. Neurologic complications of HIV disease and their treatment. Topics in HIV medicine: a publication of the International AIDS Society, USA. 2009;17(2):46.
4. Thurnher MM, Post MJ. Neuroimaging in the brain in HIV-1 infected patients. Neuroimaging Clin. 2008;18(1):93-117.
5. Yousry TA, Major EO, Ryschkewitsch C, Fahle G, Fischer S, Hou J, et al. Evaluation of patients treated with natalizumab for progressive multifocal leukoencephalopathy. New England J Med. 2006;354(9):924-33.
6. McCombe JA, Auer RN, Maingat FG, Houston S, Gill MJ, Power C. Neurologic immune reconstitution inflammatory syndrome in HIV/AIDS Outcome and epidemiology. Neurol. 2009;72(9):835-41.
7. Crum-Cianflone N, Hullsiek KH, Satter E, Marconi V, Weintrob A, Ganesan A, et al. Cutaneous

malignancies among HIV-infected persons. Archives of internal medicine. 2009;169(12):1130-8.
8. Descamps M, Hyare H, Zerizer I, Ger HJ. Neuroimaging of CNS involvement in HIV. J HIV Ther. 2008;13(3):48-54.
9. Smith AB, Smirniotopoulos JG, Rushing EJ. Central nervous system infections associated with human immunodeficiency virus infection: radiologic-pathologic correlation. Radiograph. 2008;28(7):2033-58.
10. Allen LM, Hasso AN, Handwerker J, Farid H. Sequence-specific MR imaging findings that are useful in dating ischemic stroke. Radiograph. 2012;32(5):1285-97.
11. Donnan GA, Baron JC, Ma H, Davis SM. Penumbral selection of patients for trials of acute stroke therapy. Lancet Neurol. 2009;8(3):261-9.
12. Harris AD, Coutts SB, Frayne R. Diffusion and perfusion MR imaging of acute ischemic stroke. Magnetic Resonance Imaging Clin. 2009;17(2):291-313.
13. Mark VW, Taub E, Perkins C, Gauthier LV, Uswatte G, Ogorek J. Poststroke cerebral peduncular atrophy correlates with a measure of corticospinal tract injury in the cerebral hemisphere. Am J Neuroradiol. 2008;29(2):354-8.
14. Sinha S, Sharma BS. Neurocysticercosis: a review of current status and management. J Clin Neurosci. 2009;16(7):867-76.
15. Martinez JV, Mazziotti JV, Efron ED, Bonardo P, Jordan R, Sevlever G, et al. Immune reconstitution inflammatory syndrome associated with PML in AIDS: a treatable disorder. Neurol. 2006;67(9):1692-4.
16. Raininko R, Elovaara I, Virta A, Valanne L, Haltia M, Valle SL. Radiological study of the brain at various stages of human immunodeficiency virus infection: early development of brain atrophy. Neuroradiol. 1992;34(3):190-6.
17. Sclafani DV, Mackay RS, Meyerhoff DJ, Norman D, Weiner MW, Fein G. Brain atrophy in HIV infection is more strongly associated with CDC clinical stage than with cognitive impairment. J Int Neuropsychol Soc. 1997;3(3):276-87.
18. 18. Stubbe-Drger B, Deppe M, Mohammadi S, Keller SS, Kugel H, Gregor N, Evers S, Young P, Ringelstein EB, Arendt G, Knecht S. Early microstructural white matter changes in patients with HIV: a diffusion tensor imaging study. BMC neurology. 2012 Dec;12(1):23.
19. Zhu T, Zhong J, Hu R, Tivarus M, Ekholm S, Harezlak J, et al. Patterns of white matter injury in HIV infection after partial immune reconstitution: a DTI tract-based spatial statistics study. J Neurovirol. 2013;19(1):10-23.
20. Engsig FN, Hansen ABE, Omland LH, Kronborg G, Gerstoft J, Laursen AL, et al. Incidence, clinical presentation, and outcome of progressive multifocal leukoencephalopathy in HIV-Infected patients during the highly active antiretroviral therapy era: a nationwide cohort study. J Infect Dis. 199(1):77-83.
21. Berger JR, Pall L, Lanska D, Whiteman M. Progressive multifocal leukoencephalopathy in patients with HIV infection. J Neurovirol. 1998;4(1):59-68.
22. Thurnher MM, Rieger A, Kleibel-Popov C, Settinek U, Henk C, Haberier C. Primary central nervous system lymphoma in AIDS: a wider spectrum of CT and MRI findings. Neuroradiol. 2001;43(1):29-35.
23. Slone HW, Blake JJ, Shah R, Guttikonda S, Bourekas EC. CT and MRI findings of intracranial lymphoma. Am J Roentgenol. 2005;184(5):1679-85.
24. Newell ME, Hoy JF, Cooper SG, DeGraaff B, Grulich AE, Bryant M, et al. Human immunodeficiency virus: related primary central nervous system lymphoma. Cancer. 2004;100(12):2627-36.
25. Ammassari A, Scoppettuolo G, Murri R, Pezzotti P, Cingolani A, Del CB, et al. Changing disease patterns in focal brain lesion-causing disorders in AIDS. Journal Acquired Immune Deficiency Syndromes Human Retrovirol. 1998;18(4):365-71.
26. Luft BJ, Hafner R, Korzun AH, Leport C, Antoniskis D, Bosler EM, et al. Toxoplasmic encephalitis in patients with the acquired immunodeficiency syndrome. New England J Med. 1993;329(14):995-1000.
27. Simone IL, Federico F, Tortorella C, Andreula CF, Zimatore GB, Giannini P, et al. Localised 1H-MR spectroscopy for metabolic characterisation of diffuse and focal brain lesions in patients infected with HIV. J Neurol Neurosurg Psych. 1998;64(4):516-23.
28. Chang L, Miller BL, McBride D, Cornford M, Oropilla G, Buchthal S, Chiang F, Aronow H, Beck CK, Ernst T. Brain lesions in patients with AIDS: H-1 MR spectroscopy. Radiology. 1995 Nov;197(2):525-31.
29. Tipping B, de Villiers L, Wainwright H, Candy S, Bryer A. Stroke in patients with human immunodeficiency virus infection. J Neurol Neurosurg Psych. 2007;*78*(12):1320-4.
30. Ortiz G, Koch S, Romano JG, Forteza AM, Rabinstein AA. Mechanisms of ischemic stroke in HIV-infected patients. Neurol. 2007;68(16):1257-61.
31. Miszkiel KA, Hall-Craggs MA, Miller RF, Kendall BE, Wilkinson ID, Paley MN, et al. The spectrum of MRI findings in CNS cryptococcosis in AIDS. Clinical Radiol. 1996;51(12):842-50.
32. Tien RD, Chu PK, Hesselink JR, Duberg A, Wiley C. Intracranial cryptococcosis in immunocompromised patients: CT and MR findings in 29 cases. Am J Neuroradiol. 1991;12(2):283-9.
33. Parija SC, Gireesh AR. A serological study of cysticercosis in patients with HIV. Revista do Instituto de Medicina Tropical de São Paulo. 2009;51(4):185-9.

34. Foyaca-Sibat H, Ibañez-Valdés LD, Awotedu A, Fernandez-Mena C. Neurocysticercosis in the former Transkei. In 7th Internet World Congress for Biomedical Sciences. Inabis. 2002:14-20.
35. Levy RM, Bredesen DE, Rosenblum ML. Neurological manifestations of the acquired immunodeficiency syndrome (AIDS): experience at UCSF and review of the literature. J Neurosurg. 1985;62(4):475-95.
36. Ammassari A, Cingolani A, Pezzotti P, De Luca A, Murri R, Giancola ML, et al. AIDS-related focal brain lesions in the era of highly active antiretroviral therapy. Neurol. 2000;55(8):1194-200.

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