Computer Tomographic Findings of the Brain in HIV-Patients at Ramathibodi Hospital

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Objective: *To determine the underlying cause of the brain lesions in adult HIV patients referred for CT scan at Ramathibodi Hospital and to evaluate accuracy of CT for the diagnosis of the brain lesion.*
Material and Method: *Data from first CT scan of the brain of 195 adult HIV patients at Ramathibodi Hospital were reviewed. The final diagnoses from medical records were assessed followed by CSF analysis, pathological report, and therapeutic treatment. The accuracy of the CT brain was evaluated using sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV).*
Results: *One hundred ninety five adult seropositive patients for HIV underwent CT scan of the brain, 59% were HIV encephalopathy (HIVE), 22% toxoplasmosis, 9% cryptococcoma, 5% tuberculous meningitis, 4% tuberculoma, 3% progressive multifocal leukoencephalopathy (PML), 2% lymphoma, and 1% normal. In non-specified causes (from CT scan), 33% were meningitis, 4% cerebritis, and 5% infarction. CT was found to have high sensitivity, specificity, PPV, and NPV for toxoplasmosis (92%, 96%, 86%, and 98%, respectively).*
Conclusion: *HIV encephalopathy was the most common finding of adult HIV brains. Toxoplasmosis was the most common opportunistic parenchymal brain lesion in adult HIV brains. CT was the modality of choice for diagnosis and exclusion of toxoplasmosis, but it cannot determine the cause of disease showing meningitis pattern.*

Keywords: *Computer tomography, Brain, HIV-patients*

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First described in June 1981, acquired immunodeficiency syndrome (AIDS) continues to be a worldwide major health problem. The World Health Organization estimated that 35.9-44.3 million people in the world were infected with HIV by the end of 2004[1].

Human Immunodeficiency Virus (HIV) is neurotropic and crosses the blood-brain barrier at the early stage of the disease. Thus, the central nervous system (CNS) is a major target of HIV, with approximately two-thirds of patients developing CNS involvement during the course of their disease. The spectrum of CNS diseases in AIDS can be broadly categorized into primary effect of HIV, opportunistic infections, neoplasms and vascular diseases[2,3]. The prevalence of neurological disease in symptomatic HIV-infected patients has been estimated to be 39%-70%[4–7]. In a recent review of 390 autopsies of AIDS patients, abnormal pathologies of the brain were found in 63% including cryptococcal meningitis, fulminant bacterial meningitis, neurotuberculosis, toxoplasmosis, and neurosyphilis[8,9].

Clinical presentation in HIV-CNS is non-specific. The T-helper/ inducer count (CD 4 count) may help predict which group of HIV gets CNS disease. However, it is also non-specific[10-13]. Further investigation is needed to determine the definite etiology. Standard tests for cerebrospinal fluid (CSF) include determinations of CSF protein, sugar and cell count, but these results are also non-specific. CSF serology specific antigen, CSF culture, and Polymerase Chain Reaction (PCR) have high specificity (90-100%), but the sensitivity is only about 56-60%, and is not widely

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available[7]. Brain biopsy is a reference standard modality[7]. However, it is an invasive procedure that places the surgical personnel at risk of occupational infection of HIV. Therefore, the use of this procedure is limited[14-16].

Diagnostic imaging still relies on radiological interpretation. Imaging modalities include Computer Tomography (CT), Magnetic Resonance Imaging (MRI), Single-Photon Emission Computed Tomography (SPECT), and Positron Emission Tomography (PET). They may assist in confirmation of diagnosis and selection of treatment protocol. MRI is the modality of choice, with a high sensitivity in lesion detection, but the availability is limited and the cost is high. MRI is usually performed when CT findings are inconclusive. Imaging patterns of brain lesions in HIV patients can be divided into three groups: negative findings, atrophic brain, and focal lesion[17,18]. SPECT and PET are nuclear medicine imaging modalities that can be performed to differentiate malignant lesions from non-malignant lesions. However, they are non-specific for determining the cause. Moreover, technologies are not yet widely available and still expensive[2,16-18].

CT scan has revolutionized the assessment of patients presenting with neurologic symptoms. Although it is less sensitive than MRI, CT is still the preferred first line imaging modality for detecting brain lesions and screening prior to lumbar puncture in HIV patients due to its wide availability, non-complicated technique, and relatively low cost[2,16].

Several studies of imaging in HIV-CNS have been performed in the past. The results vary in disease and incidence in each geographic area[19-21]. The primary purpose of the present study was to determine the cause of brain lesions in adult HIV patients referred for CT scan at Ramathibodi Hospital from January 2001 to December 2005. The secondary purpose was to evaluate sensitivity, specificity, positive predictive value, and negative predictive value of CT for diagnosis of brain lesion.

Material and Method

Patients

This was a retrospective study, approved by our institutional research ethics committee. The authors reviewed our database from the Advance Diagnostic Imaging and Image-guided Minimal Invasive Therapy Center (AIMC), Ramathibodi Hospital, for adult HIV patients who underwent CT scan of the brain, from January 2001 to December 2005. Patients were included in the present study if they were seropositive for HIV, age 18-80 years with first CT brain examination at Ramathibodi Hospital. The exclusion criteria included patients with missing or inadequate data.

CT imaging technique

The non-contrast and contrast axial CT scans were obtained with Light Speed Plus (General Electric Medical System, Milwaukee, Wis., USA,) using 2.5 mm slice thickness at the posterior fossa and 7.5 mm of the rest of the brain from January 2001 to July 2005, and Somatom Sensation Cardiac 64, (Siemens, Germany) using 3.0 mm slice thickness of the whole brain from August 2005 to December 2005. Intravenous non-ionic contrast material was administered, using average dose 1ml/kg of 300 mg iodine /ml and contrast axial study was performed immediately after manual injection of contrast media.

Imaging interpretation

The CT diagnoses were interpreted by two participants, one is a board certified neuroradiologist (JL) and another one is a third year radiology resident (KH). Both of whom were blinded to the clinical details.

Typical findings of each disease were referenced[2,4,6,10]. HIV encephalopathy is defined as atrophic brain advanced more than age and/or symmetrical multifocal hypodense area in the cerebral deep white matter. Toxoplasmosis is defined as single or multiple rim/nodular enhancing, hypo/isodense lesion with perilesional edema and mass effect at basal ganglion and/or cerebral hemisphere. Cryptococcoma is defined as non-enhancing, low-density lesion without associated perilesional edema along basal ganglia and/or perivascular space. Progressive multifocal leukoencephalopathy (PML) is defined as asymmetrical focal non-enhancing, hypodense white matter lesion without mass effect. Tuberculosis is defined as ring/nodular/irregular enhancing iso/hypodense/slightly hyperdense lesion and/ or target lesion as well as basal cistern leptomeningeal enhancement. Lymphoma is defined as homogeneous or rim- enhancing, hyper or hypodense lesion with perilesional edema at periventricular region. Herpes simplex encephalitis is defined as patchy of gyriform enhancing, low-density lesion at temporal lobe with mild mass effect. Meningitis is defined as leptomeningeal enhancement. Cerebritis is defined as ill-defined enhancing, poorly marginated subcortical hypodense area. Infarction is defined as non-enhancing or gyriform enhancing, ill or well-defined hypodene lesion of vascular territory or perforating substance. Normal brain is defined as negative finding.

Lesion confirmation

The final diagnosis from medical records was assessed, followed by ICD 9 or 10, based on history, neurological examination, laboratory data, and therapeutic treatment (including one or more method in each patient). Positive laboratory data were included CSF culture, CSF-specific antigen, CSF-PCR, CSF india-ink (for cryptococcus), CSF cytology, blood for antigen, and pathological report.

Statistic analysis

All analyses were performed using software (STATA, Version 8.0). The geographic and CT imaging data were presented as mean, range, frequency and percentage. The statistical values of the CT brain were evaluated using sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV).

Results

Two hundred forty eight patients sero-positive for HIV underwent CT brain study at Ramathibodi Hospital between January 2001 and December 2005. Fifty-three of them were excluded for missing or inadequate data. Consequently, the study population included 195 patients with 114 males and 81 females. The mean age was 34 years (range 21-77 years). One hundred ninety patients had neurologic symptoms. Most common presenting neurologic symptom was headache (42%). Five patients had non-neurologic symptoms, of which dyspnea was the presenting symptom. The mean absolute lymphocyte count (ALC) was 864 cell/mm^3 (range 17-4403 cell/mm^3). Only 158 patients had serum CD4 count whose mean was 49 cell/mm^3 (range 1-747 cell/mm^3). Details of the patient data are shown in Table 1.

All patients, except in two cases, had abnormal CT findings. The most common CT diagnoses were HIV-encephalopathy (HIVE) in 144 patients (59%). All of them were atrophic brain [Fig. 1] and some of them had multifocal hypodense deep white matter lesion (50 patients) [Fig. 2, Table 2]. Toxoplasmosis was diagnosed by CT in 42 patients (22%), while the final diagnosis was confirmed in 39 patients who were proven by 23 therapeutic treatments and 16 positive CSF/blood tests [Table 3]. The uncorrected diagnoses by CT in three cases were proven by therapeutic treatment to be tuberculoma [Fig. 3]. Most common CT scan finding of toxoplasmosis were multiple deep and superficial rim-enhancing lesions located in both cerebral hemispheres and basal ganglia associated with edema and mass effect. At presentation there was 76% multiple lesions and 24% single lesions [Table 2, Fig. 4, 5, 6]. The sensitivity, specificity, PPV, and NPV of CT for diagnosis toxoplasmosis were 92.3%, 96.2%, 85.7%, and 98.0%, respectively [Table 3]. Cryptococcoma were diagnosed in 17 patients (9%). True positive of this disease was 15 in 17 patients [Table 3]. Two cases were proven to be lacunar infarction by therapeutic treatment [Fig. 7 and 8]. Most often, CT scans findings were non-enhancing hypodense lesions at the basal ganglion [Table 2, Fig. 9]. Progressive multifocal leucoencephalopathy (PML) was diagnosed in six patients (3%), while final diagnosis was in seven patients [Table 2]. There was one case of PML, which proved by CSF-PCR, but CT was diagnosed to be infarction [Fig. 10]. CT findings of PML were non-enhancing hypodense white matter lesion without mass effect. Most common location was parietal and/or occipital lobes [Fig. 11].

Table 1. 195 baseline patient characteristics

Patient characteristics	Number (%)
Sex	
Male	114 (58.5)
Female	81 (41.5)
Age group (mean 34 yrs, range 21-77 yrs)	
18-35	112 (57.4)
36-53	70 (35.9)
≥ 54	13 (6.7)
Symptoms	
Headache	81 (41.5)
Alteration of consciousness	65 (33.3)
Seizure	23 (11.8)
Mortor weakness	19 (9.7)
Other neurogenic symptom	2 (1.0)
Non-neurologic symptom	5 (2.6)
White blood count (WBC)	
(mean 5,130 cell/mm^3, range 1,050-20,200 cell/mm^3)	
< 3,000	23 (11.8)
3,000-10,000	148 (75.9)
> 10,000	24 (12.3)
Absolute lymphocyte count (ALC)	
(mean 864 cell/mm^3, range 17-4,403 cell/mm^3)	
< 2,000	171 (87.7)
≥ 2,000	24 (12.3)
T-helper/ inducer count (CD 4)	
(mean 49 cell/mm^3, range 1-747 cell/mm^3) (n = 158)	
< 200	126 (79.8)
200-500	25 (15.8)
> 500	7 (4.4)

Note: normal WBC = 4,000-10,000 cell/mm^3
normal ALC = 1000-3,500 cell/mm^3
normal CD4 = 500-1,500 cell/mm^3

Table 2. CT finding of the disease

Etiology	CT diagnosis (No.)	CT pattern [n]													
		Non-enhanced				No. of lesion		Mass effect	Contrast enhanced				Lepto.	Hydrocephalus	
		A	HP	IS	HPO	S	M		NE	HE	HeE	RE		OB	Com
HIVE	114	114a	-	-	-	-	-	-	-	-	-	-	-	-	-
Toxoplasmosis	42b	-	-	9	39	10	32	41	-	6	3	33	-	7	-
Cryptococcoma	17	-	-	2	15	4	13	2	17	-	-	-	-	-	10
PML	6c	-	-	-	6	2	4	2	6	-	-	-	2	1	-
Tuberculoma	8*	-	2d	1	5	2	6	8	-	3	1	4e	-	1	2
Tuberculous meningitis	10*	-	-	-	-	-	-	-	-	-	-	-	10f	-	4
Lymphoma	4	-	4	-	-	2	2	4	-	4	-	-	3	-	-
Herpes	2g	-	-	-	2	2	-	2	-	1	1	-	-	-	-
Meningitis^	63	-	-	-	-	-	-	-	-	-	-	-	63	-	40
Cerebritis	7	-	-	-	7	5	2	3	-	3	4	-	3	1	-
Infarction	11	-	-	-	11	3	7	-	11	-	-	-	-	-	-

A = atrophic; HP = hyperdense; IS = isodense; HPO = hypodense; S = single; M = multiple; NE = non-enhancement; HE = homogeneous enhancement; HeE = heterogeneous enhancement; RE = rim-enhancement; Lepto. = leptomeningeal enhancement; OB = obstructive; Com = communicating

^ = unspecified cause

a = 50 of 114 have multifocal hypodense cerebral deep white matter

b = cerebral hemisphere and basal ganglia in all case

c = white matter lesion

d = calcific foci

e = target lesion in 2 cases

f = basal cistern leptomeningeal enhancement

g = temporal lobe lesion

* = combine disease (8 cases of tuberculous meningitis have tuberculoma)

Table 3. CT, Final diagnoses and statistic evaluation of CT brain for disease diagnosis

Etiology	CT diagnosis (No.)	Final diagnosis (No.)	Evidence support			CT/Final diagnosis				CT value			
			Positive CSF/blood (No.)	Biopsy (No.)	Therapeutic treatment (No.)	TP (No.)	FP (No.)	TN (No.)	FN (No.)	Sensitivity (95%CI)	Specificity (95%CI)	PPV (95%CI)	NPV (95%CI)
Normal brain	2	1#	-	-	-	1	1	192	1	50 (43-57)	99.5 (98-100)	50.0 (43-57)	99.5 (98-100)
HIVE	114	105	*	-	*	102	12	78	3	97.1 (95-99)	86.7 (82-91)	89.5 (85-94)	96.3 (94-99)
Toxoplasmosis	42	39	16a	-	23	36	6	150	3	92.3 (89-96)	96.2 (93-99)	85.7 (81-91)	98.0 (96-100)
Cryptococcoma	17	15	12b	-	3	15	2	178	0	100.0 (100)	98.9 (97-100)	88.2 (85-91)	100.0 (100)
PML	6	7	4c	-	3	5	1	182	2	71.4 (65-78)	99.5 (98-100)	83.3 (78-89)	98.9 (97-100)
Tuberculoma	8	12	8d	-	4	8	0	183	4	66.7 (60-75)	100.0 (100)	100.0 (100)	97.8 (95-100)
Tuberculous meningitis	10	9	10e	-	-	9	1	185	0	100.0 (100)	99.4 (98-100)	90.0 (86-94)	100.0 (100)
Lymphoma	4	2	-	2	-	2	2	191	0	100.0 (100)	(97-100)	50.0 (43-57)	100.0 (100)
Herpes	2	0	-	-	-	0	2	193	0	*	*	*	*
Meningitis	63	78^	76f	-	2	53	10	107	25	67.9 (62-74)	91.4 (88-94)	84.1 (79-89)	81.1 (76-86)
Cerebritis	7	0	-	-	-	0	7	188	0	*	*	*	*
Infarction	11	5	-	-	5	5	6	184	0	100.0 (100)	96.8 (94-99)	45.5 (38-52)	100.0 (100)

* = not assessment; TP = true positive; FP = false positive; FN = false negative; TN = true negative; PPV = positive predictive value; NPV = negative predictive value
^ = TB meningitis + cryptococcal meningitis + meningitis (unspecified cause)
a = 15 blood antigen and 5 CSF-antigen
b = 12 CSF-culture, 3 india-ink
c = 4 CSF-PCR
d = 8 CSF-culture, 2 CSF-PCR
e = 10 CSF-culture, 2 CSF-PCR, 1 CSF-antigen
f = 48 CSF-culture for cryptococcus, 20 india-ink, 28 CSF-culture for TB, 10 CSF-PCR for TB
# = cryptococcal meningitis in 1 case (by CSF-culture and India ink)

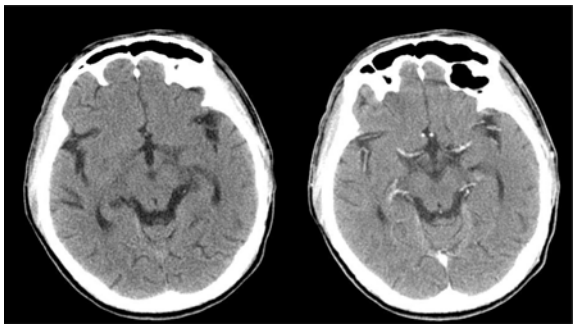

Fig. 1 HIVE; diffuse brain atrophy advance than age in 29 year-old patient, whom has positive serum anti-HIV

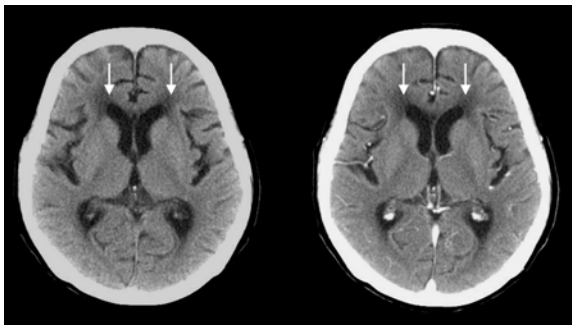

Fig. 2 HIVE; 30-year-old HIV patient shows diffuse brain atrophy advance than age with symmetrical hypodense area at deep white matter of both frontal lobes (arrows)

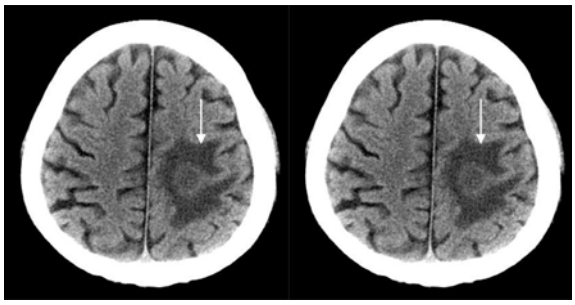

Fig. 3 1 in 3 cases of uncorrected diagnosis toxoplasmosis. A 35-year-old HIV patient shows single rim-enhancing isodense lesion with perilesional edema at the left frontoparietal lobe (arrow), representing toxoplasmosis by CT. In final diagnosis was tuberculoma proved by therapeutic treatment

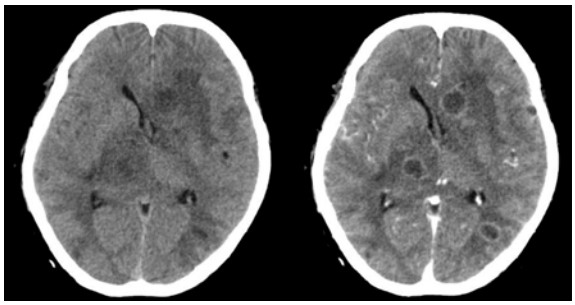

Fig. 4 Toxoplasmosis; multiple rim-enhancing lesions with perilesional edema at the left basal ganglion, right thalamus, left frontal and occipital lobes. Associated mass effect is seen

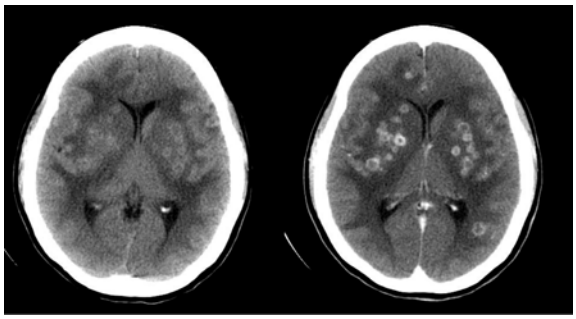

Fig. 5 Another case of toxoplasmosis; multiple small enhancing lesions with perilesional edema. Final diagnosis was proved to be toxoplasmosis by positive CSF-antigen for toxoplasma

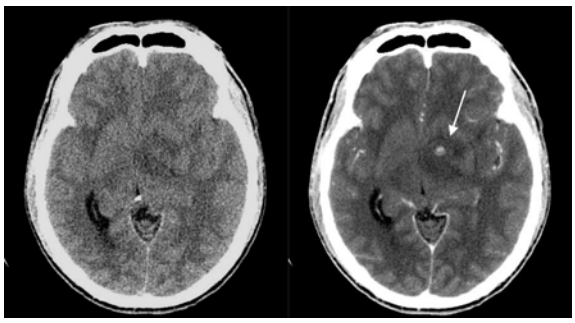

Fig. 6 Another case of toxoplasmosis; small nodular enhancing lesion with perilesional edema at left basal ganglion (arrow). Final diagnosis was proved to be toxoplasmosis by therapeutic treatment

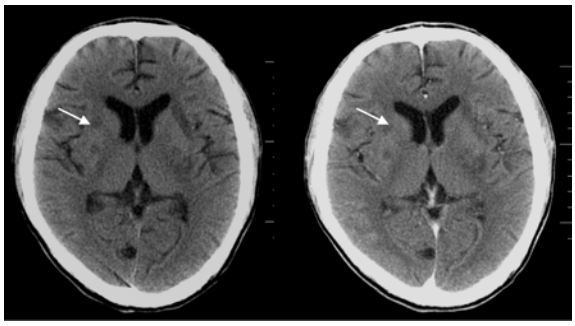

Fig. 7 First cases of uncorrected diagnose cryptococcoma by CT; few small non-enhancing hypodense lesions at the right basal ganglion (arrow), likely to be cryptococcoma by CT. Final diagnosis was diagnosed to be lacunar infarction by therapeutic treatment. Other hypodense lesions at left basal ganglion and right occipital lobe were subacute and old infarction, respectively

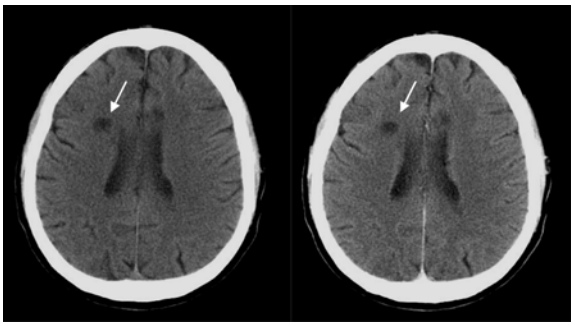

Fig. 8 Second cases of uncorrected diagnose cryptococcoma by CT; few small non-enhancing hypodense lesions at both frontal lobe (arrow), likely to be cryptococcoma by CT. Final diagnosis was diagnosed to be lacunar infarction by therapeutic treatment

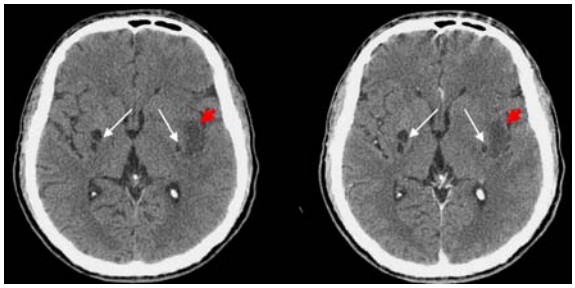

Fig. 9 Cryptococcoma; few non-enhancing well defined hypodense lesions at basal ganglia (arrows) representing cryptococcomas. Incidental subacute infarction is seen at the left insular cortex (arrowhead)

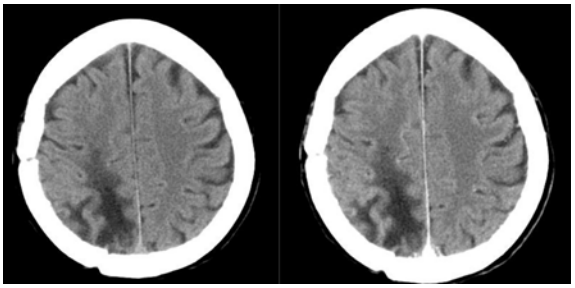

Fig. 10 PML; in this case, territory infarction of right anterior cerebral artery (ACA) was diagnosed by CT. Final was proved to be PML by positive CSF-PCR for papova virus

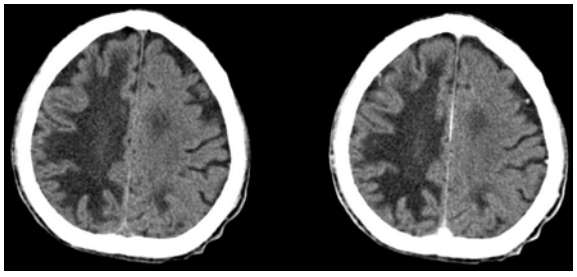

Fig. 11 PML; non-enhancing hypodense lesion involving right frontoparietooccipital and left parietal lobes without mass effect

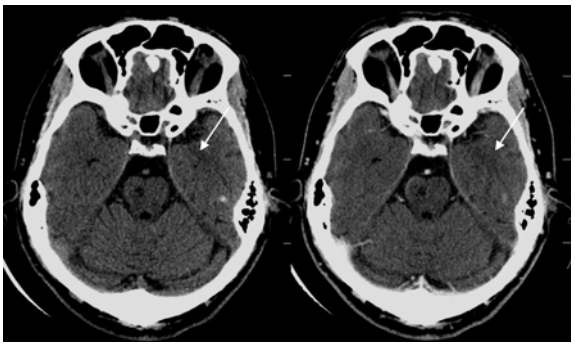

Fig. 12 Uncorrected diagnose herpes simplex encephalitis; CT shows slightly enhancing ill-defined hypodense area at the left temporal lobe (arrow), could be herpes simplex encephalitis. Final diagnosis was tuberculoma by therapeutic treatment. Note old lacunar infarction at right the paramedian pons

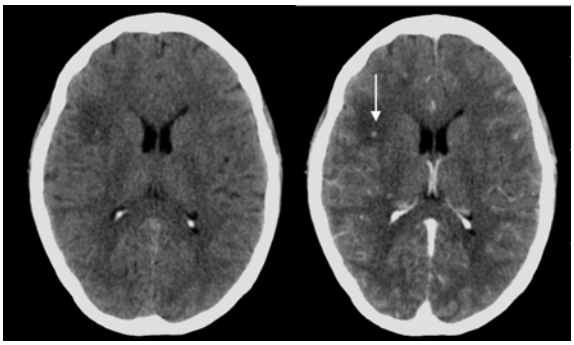

Fig. 13 Tuberculoma; nodular-enhancing with perilesion edema at the right frontal lobe (arrow). Note diffuse leptomeningeal enhancement. Final was proved by positive CSF culture

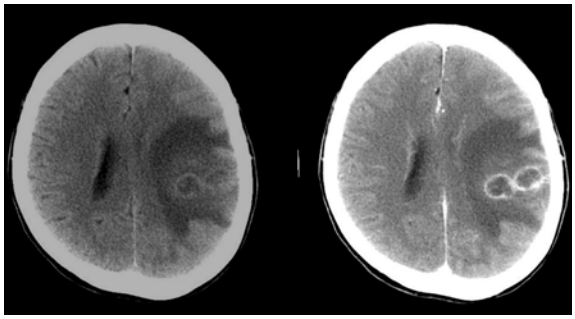

Fig. 14 Tuberculoma; rim-enhancing with central hyperdense lesions and perilesion edema at the left parietal lobe. Note that the patient has co-pulmonary tuberculosis

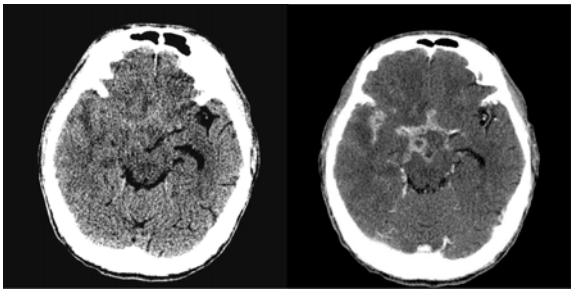

Fig. 15 1 in 10 cases of tuberculous meningitis by CT; marked leptomeningeal enhancement at basal cistern. Final was proved by CSF culture and PCR

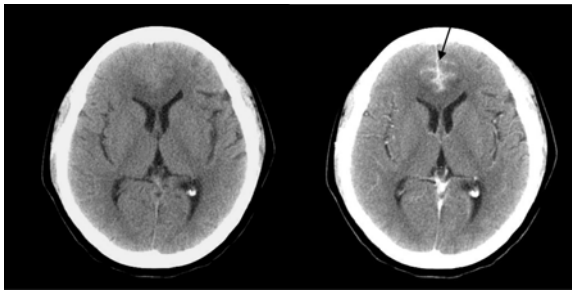

Fig. 16 Another case of tuberculous meningitis by CT; marked leptomeningeal enhancement at subarachnoid space of the frontal lobe (arrow). Final was proved by CSF culture and PCR

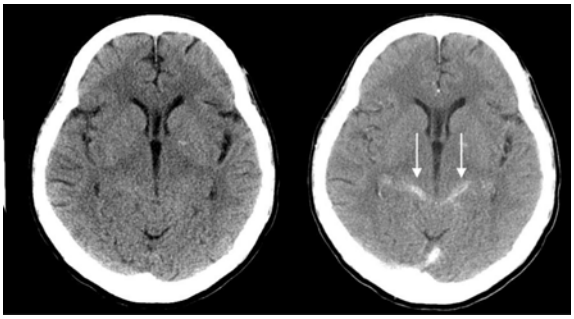

Fig. 17 A 25-year-old patient was diagnosed tuberculous meningitis by CT due to leptomeningeal enhancement at bilateral basal cisterns (arrows). However, final was proved by CSF culture and positive india ink for cryptococcosis

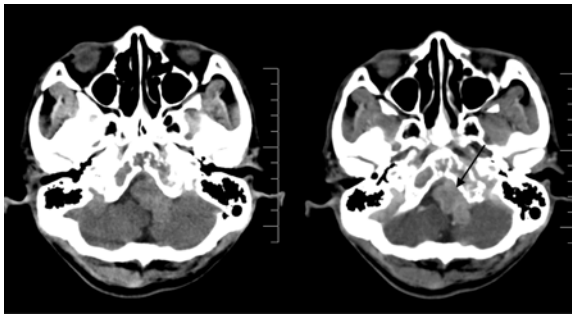

Fig. 18 A 40-year-old patient was diagnosed lymphoma by CT (arrow), however, final was proved by pathological report to be smooth muscle cell tumor

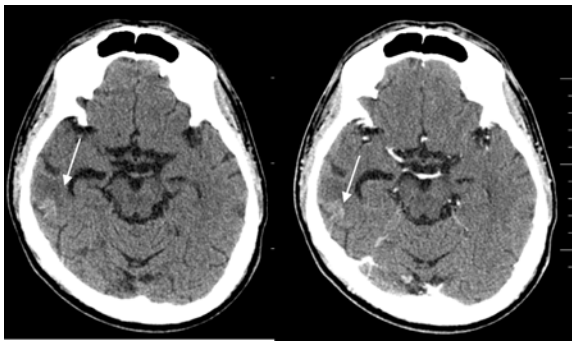

Fig. 19 Another case of uncorrected diagnosed lymphoma by CT. Final diagnosis was proved by pathological report to be chronic inflammation

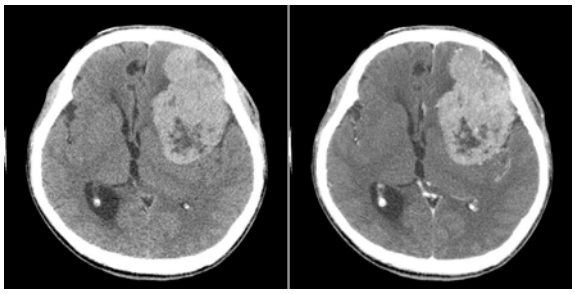

Fig. 20 Malignant lymphoma; homogeneous enhancing intra/extra-axial hyperdense mass with internal hypodense lesions and mass effect. Biopsy was proved in lymphoma

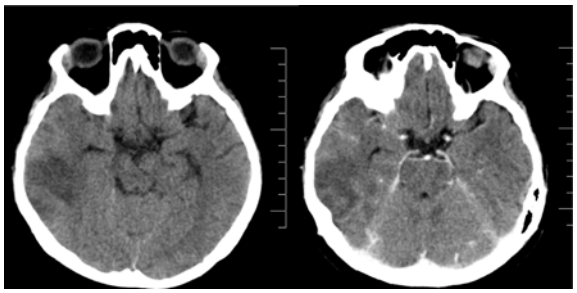

Fig. 21 Another case of uncorrected diagnosed herpes simplex encephalitis; CT shows gyriform enhancing ill-defined hypodense area at the right temporal lobe, could be herpes simplex encephalitis. Final diagnosis was toxoplasma encephalitis by therapeutic treatment

Its CT sensitivity and specificity were 71.4% and 99.5% [Table 3]. Tuberculoma was diagnosed in eight patients (4%) by CT, in whom there were 100% true positive. However, there were 12 patients in final diagnosis (3 cases from toxoplasmosis and one case from herpes simplex encephalitis) by CT therapeutic treatment [Fig. 3 and 12, Table 3]. CT pattern were ring/nodular enhancing patterns with perilesional edema [Fig. 13]. Target lesion was found in two patients [Fig. 14, Table 2] and enhanced basal cistern was found in eight cases [Fig. 15 and 16, Table 2]. There were 10 cases of tuberculous meningitis by CT [Table 2 and 3, Fig. 15 and 16], which proved true positive in nine cases and false positive in one case with cryptococcal meningitis as final diagnosis [Fig. 17]. Four cases of lymphoma were diagnosed by CT, but final diagnosis was summarized in two cases, and confirmed by pathology report [Table 3]. The pathological reports of other two cases were smooth muscle cell tumor [Fig. 18] and chronic inflammation [Fig. 19]. CT pattern had 100% hyperdense and homogeneous enhancement mass [Table 2]. Most common location was periventicular region. However, one patient had combined intra/extraaxial mass [Fig. 20]. Herpes simple encephalitis was diagnosed in two cases, but they were not confirmed at the final diagnosis [Table 2], which was found to be toxoplasmosis in one case and TB in one case [Fig. 21 and 12], by therapeutic treatment. In non-specified cause, meningitis was most commonly diagnosed by CT in 63 patients (32%). Most common CT findings were communicating hydrocephalus and leptomeningeal enhancement [Table 2, Fig. 22]. The sensitivity, specificity, PPV, and NPV of CT were 67.9%, 91.4%, 84.1%, and 81.1%, respectively [Table 3]. The final diagnoses of meningitis were found in 78 patients caused by cryptococcosis, TB and non-specific. However, in 63 patients from CT diagnoses, seven (11.1%), 11(17.5%), six (9.5%), two (3.2%) and one (1.6%) were proven for HIVE, cryptococcosis, tuberculous meningitis, non-specified meningitis and others such as tension headache, respectively. Combined diagnoses of HIVE + cryptococcosis were 22 (34.9%), HIVE + TB 12 (19.1%) and HIVE + other two (3.2%). Cerebritis was diagnosed in seven cases by CT, but not confirmed in the final diagnosis. In the present study, Infarction was diagnosed in 11 cases by CT, but only five cases were confirmed in final diagnosis as evidenced by therapeutic treatment. Another six cases were meningitis in final diagnosis (TB 3 cases and cryptococcosis 3 cases [Fig. 23]). Its CT finding showed non-enhancing hypodense lesion [Fig. 24,Table 2]. The sensitivity and specificity were 100% and 96.8% [Table 3].

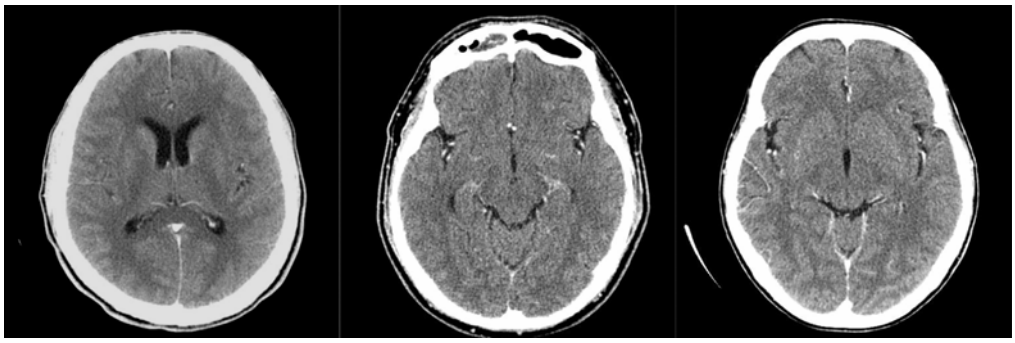

Fig. 22 Meningitis by contrast enhanced CT; diffuse leptomeningeal enhancement in cryptococcal (a), tuberculous (b) and non-specified cause (c) meningitis, respectively. Note positive CSF for cryptococcus and TB in figure a and b, respectively

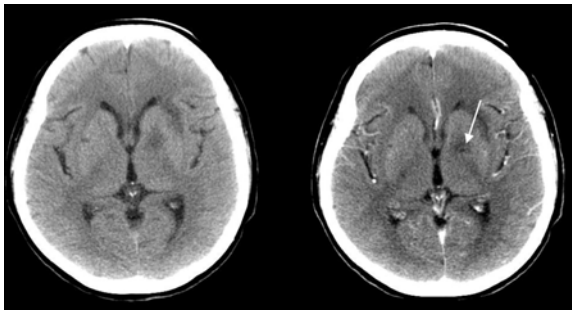

Fig. 23 Acute lacunar infarction at genu of the left internal capsule and left gobus pallidus by CT (arrow). Final diagnosis was proved by positive CSF for cryptococcosis. Note old lacunar infarction at right basal ganglion and posterior limb of the right internal capsule

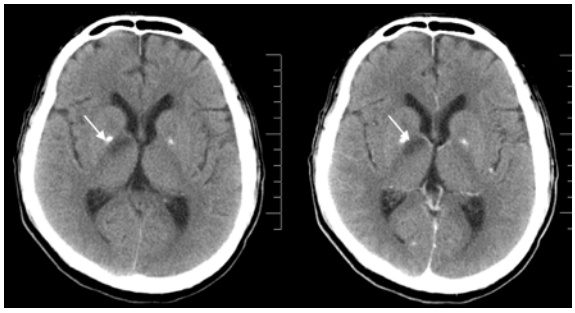

Fig. 24 Acute lacunar infarction at posterior limb of the right internal capsule by CT (arrow). Final diagnosis was proved by clinical and therapeutic treatment to be infarction. Patient was presented with grade II left hemiparesis

Discussion

The increasing number of AIDS patients and the high incidence of neurologic manifestation in this syndrome have led to a rapid increase in the frequency of neuroradiologic evaluation in patients with AIDS. In this study, the CT brain was diagnosed, including 59.1% were HIV encephalopathy (HIVE), 21.8% toxoplasmosis, 8.8% cryptococcoma, 5.0% tuberculous meningitis, 4.2% tuberculoma, 3.1% progressive multifocal leukoencephalopathy (PML), 2.1% lymphoma, 1.0% herpes simplex encephalitis and 1% normal, respectively. In non-specified cause, meningitis is diagnosed in 32.6%. The rest were 5.2% infarction and 3.6% cerebritis. There were several studies about HIV brain lesions such as the study of Ammassari et al[15] that reviewed brain imaging of 281 HIV patients in 1991-1998. The result showed toxoplasmic encephalitis 36.4%, CNS lymphoma 26.7%, PML 18.2%, HIVE 5%, mycotic abscess 2.7% and tuberculoma 5%. The study of Steinmetz H et al[20] is another study that reviewed 188 seropositive patients in 1988-1991. They reported toxoplasmosis 25%, HIVE 10.1%, PML 4.8%, cerebral lymphoma 0.5% and other conditions in 4.8%. From the two previous reports and the present findings, the results are varying in disease and incidence, and the reason may be from difference geographic area and period of evaluation. However, toxoplasmosis was found to be the most parenchymal opportunistic infection in HIV brain patients.

Toxoplasmosis encephalitis (TE) is the most common opportunistic infection causing encephalitis or focal cerebral lesions. About 3% to 40% of patients

with AIDS will develop TE. Geographical differences in TE rates among patients with AIDS may be explained by the worldwide observed variation of Toxoplasma IgG seroprevalence[22]. A characteristic pattern often seen is multiple ring and/or nodular enhancing lesions at the corticomedullary junction and the basal ganglia with edema and mass effect [Table 2, Fig. 4, 5, 6]. In the present study, there was high PPV and NPV for the diagnosis [Table 3]. The authors suggest that CT should be used for diagnosis and exclusion of the possibility of disease. In cryptococcoma, the CT pattern shows multifocal basal ganglia and midbrain hypodense lesions, which represent both gelatinous pseudocysts and dilated perivascular spaces [Table 2, Fig. 9]. Leptomeningeal enhancement is also seen, which was meningeal involvement (most pattern of cryptococcal infection in this study). However, it could not determine underlying nature cause of meningitis form and underdiagnosed meningitis. For cryptococcoma, there was high PPV (82.2%) so that CT is still reliable for diagnosis of parenchymal form. On imaging, lacunar infarction might be miss diagnosis to be cryptococcoma (2 cases of false positive). For PML caused by the papova virus, CT characteristically shows central and/or convolutional white-matter low densities, often in a parietooccipital location and with negligible mass effect[23] [Fig. 11]. There were 83.3% PPV and 98.9% NPV; hence CT could be used for both diagnosis and exclusion of the disease. The TB shows ring/nodular enhancing patterns with perilesional edema, characteristic for tuberculoma, but not pathognomonic. Occasionally, a target sign is seen consisting of a rim-enhancing lesion with a central area of enhancement or calcification[23] [Fig. 14], which was found in two cases in the present study [Table 2]. In the present study, the tuber-culoma was 100% PPV of CT, [Table 3] because all eight cases by CT in the present study had associated finding with leptomeningeal enhancement at basal cistern, which might be helpful to diagnosis. However, in practical, when the CT scan shows only ring-enhancement pattern, it may be difficult to differentiate between toxoplasmosis and tuberculoma (three cases of toxoplasmosis and one case of herpes simplex encephalitis were false positive by CT). The authors suggest a history of pulmonary TB, favoring CNS TB infection. For tuberculous meningitis, the characteristic CT pattern shows markedly leptomeningeal enhancement at basal cistern. It was 90%PPV by CT, however, one case was proved to be cryptococcal meningitis. On CT, lymphomas have usually been described as homogeneous, heterogeneous or ring-like patterns[2]. Typical locations include the periventricular white matter, basal ganglia, corpus callosum and thalami. Uncommon locations are more common in AIDS patients such as the brain stem, cranial nerves, pineal gland and cavernous sinus [Table 2, Fig. 20]. In the present study, there was no confusion in diagnosis between toxoplasmosis and lymphoma, because all of the lymphomas in the present study showed hyperdense and homogeneous enhancement [Table 2]. The CT value showed 100% sensitivity, 99% specificity, 50% PPV and 100% NPV. The authors suggest CT should be more useful for excluding disease than diagnosis. The primary HIV infection causes HIV encephalopathy (HIVE), for which CT finding shows atrophic brain pattern more advanced than age and multifocal of hypodense deep white matter lesion [Fig. 2]. In the present study, HIVE was the most common findings in HIV adult brain and found in combination with other neurologic causes. However, final diagnosis of this disease was only proved by clinical dementia. Herpes simple encephalitis was diagnosed in two cases, but they were not confirmed at the final diagnosis [Table 2], which found to be toxoplasmosis in one case and TB in the other case [Fig. 12 and 21], by therapeutic treatment. The reason may be pattern of inhomogeneous irregular enhancement, hypodense lesion with perilesional edema and located only at the temporal lobe, favoring this disease than others.

In contradiction to parenchymal infection, meningitis infrequently appears as an abnormality on CT. Positive findings such as communicating hydrocephalus, dilated cerebrospinal fluid (CSF) spaces and meningeal enhancement are identified, but is nonspecific for underlying nature [Fig. 22]. Associated parenchymal involvement is seen infrequently. CSF analysis is, however, usually diagnostic gold standard. In the present study, when CT verified meningitis and the final diagnosis was cryptoccoccal or TB meningitis, the authors suggested that the results were true positive because the CT could not determine the cause of disease in meningitis pattern. PPV of meningitis was 84.1%, and then CT could be useful for diagnosis meningitis. Cerebrovascular disease in patients with AIDS is not commonly recognized unless the patient presents with stroke or vascular thrombosis. It is well known to occur with HIV virus, TB, cryptococcal meningitis or neurosyphilis. In the present study, infarction was diagnosed in 11 patients by CT, while final diagnosis was found in five patients by therapeutic treatment. The rest could be proved by CSF culture for TB in three cases and for cryptococcus in three cases. Furthermore, there had

combined disease between HIVE and opportunistic lesion, but had no combined disease between opportunistic and opportunistic group. Thus, there was no difficulty to evaluate the finding on CT.

The limitations of the present study include: first, rim enhancing lesion may be confused between toxoplasmosis and tuberculoma, which cause selective bias from incidence of disease; second, in meningitis pattern, CT cannot identify cause of the disease, except pattern of markedly leptomeningeal enhancement may be helpful to diagnose TB; third, CT and final diagnosis do not separate the combined disease of HIVE and opportunistic lesion in some patients so that some findings may be from HIVE; fourth, final diagnosis of toxoplasmosis and TB in some patients is not proved by laboratory data but proved by history and therapeutic treatment.

Conclusion

HIV encephalopathy was found as the most common finding of adult HIV brains. Toxoplasmosis was the most common parenchymal brain lesion in adult HIV brains. CT was found to have high sensitivity, specificity, PPV, and NPV for diagnosis of toxoplasmosis but could not determine cause displaying meningitis pattern. In some diseases that show the same findings such as toxoplasmosis and TB, history of pulmomary tuberculosis may be helpful in the diagnosis.

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การศึกษาภาพเอกซเรย์คอมพิวเตอร์สมองของผู้ป่วยเอชไอวีที่มารับการตรวจที่โรงพยาบาลรามาธิบดี

กีรติ หงษ์สกุล, จิรพร เหล่าธรรมทัศน์

วัตถุประสงค์*: การศึกษานี้แสดงลักษณะและสาเหตุของความผิดปกติที่พบในภาพเอกซเรย์คอมพิวเตอร์สมองของผู้ป่วยที่ติดเชื้อเอชไอวีที่มาตรวจที่โรงพยาบาลรามาธิบดี รวมทั้งประเมินความถูกต้องของเอกซเรย์คอมพิวเตอร์ที่ใช้วินิจฉัยความผิดปกติดังกล่าว*

วัสดุและวิธีการ*: เป็นการศึกษาย้อนหลังจากข้อมูลผู้ป่วย 195 คน โดยให้รังสีแพทย์ 2 คนแปลผลภาพเอกซเรย์คอมพิวเตอร์ก่อนโดยไม่ทราบข้อมูลเก่าจากเวชระเบียน บันทึกการอ่านฟิล์ม หรือการวินิจฉัยของผู้ป่วย จากนั้นนำมาเปรียบกับการวินิจฉัยขั้นสุดท้ายซึ่งสรุปตามข้อมูลทางคลินิก*

ผลการศึกษา*: จากภาพเอกซเรย์คอมพิวเตอร์สมองของผู้ป่วยเอชไอวี 195 คน ได้รับการวินิจฉัยว่ามีการติดเชื้อไวรัสเอชไอวีของเนื้อสมองร้อยละ 59 โปรโตซัวชนิดท็อกโสพลาสมาของเนื้อสมองร้อยละ 22 เชื้อราชนิดคริบโตค็อกคัสของเนื้อสมองร้อยละ 9 เยื่อหุ้มสมองอักเสบจากเชื้อไมโครแบคทีเรียร้อยละ 5 การติดเชื้อไมโครแบคทีเรียของเนื้อสมองร้อยละ 4 การติดเชื้อไวรัสปาโปวาของเนื้อสมองร้อยละ 3 มะเร็งชนิดลิมโฟมาร้อยละ 2 และไม่พบความผิดปกติ ร้อยละ 1 ส่วนในกลุ่มที่ไม่สามารถบอกสาเหตุที่ชัดเจนได้จากเอกซเรย์คอมพิวเตอร์ ได้รับการวินิจฉัยว่าเป็นเยื่อหุ้มสมองอักเสบชนิดไม่เจาะจงร้อยละ 33 เนื้อสมองอักเสบซีรีไบรตีสร้อยละ 4 และเนื้อสมองตายจากการขาดเลือดร้อยละ 5 การวินิจฉัยการติดเชื้อโปรโตซัวชนิดท็อกโสพลาสมาของเนื้อสมองจากภาพเอกซเรย์คอมพิวเตอร์ พบว่ามี sensitivity, specificity, positive predictive value (PPV) และ negative predictive value (NPV) เท่ากับร้อยละ 92, 96, 86 และ 98 ตามลำดับ*

สรุป*: การติดเชื้อไวรัสเอชไอวีของเนื้อสมองพบได้บ่อยที่สุด จากการตรวจเอกซเรย์คอมพิวเตอร์สมองของผู้ป่วยเอชไอวี ส่วนการติดเชื้อโรคฉวยโอกาสของเนื้อสมองที่พบบ่อยที่สุดคือโปรโตซัวชนิดท็อกโสพลาสมา การตรวจเอกซเรย์คอมพิวเตอร์สมองในผู้ป่วยเอชไอวีที่สงสัยว่าติดเชื้อโปรโตซัวชนิดท็อกโสพลาสมา มีประโยชน์ทั้งในการวินิจฉัยว่าเป็นโรคและไม่เป็นโรค อย่างไรก็ตามเอกซเรย์คอมพิวเตอร์มักไม่สามารถบอกสาเหตุที่ชัดเจนได้ในกลุ่มที่เป็นเยื่อหุ้มสมองอักเสบ*