TITLE

"CENTRAL NERVOUS SYSTEM FINDINGS AS SEEN IN PATIENTS WITH HUMAN IMMUNODEFICIENCY VIRUS INFECTION, UNDERGOING COMPUTERISED TOMOGRAPHY EXAMINATION AT KENYATTA NATIONAL HOSPITAL."

This dissertation is submitted in part fulfillment for the degree of Master of Medicine in Diagnostic Radiology of the University of Nairobi.

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Declaration

Candidate
This dissertation is my original work and has not been presented for a degree in any other university.

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DEDICATION

I would like to dedicate this thesis to the memory of my late mother Mrs Milkah Wangui Gatune, whom I lost during my post graduate course.

CONTENTS

AIM

To assess the clinical utility of cranial CT scanning in AIDS patients with neurological symptoms.

OBJECTIVES

1. **Study the CT scan findings in HIV patients undergoing cranial CT scan examination.**

2. **Correlate clinical presentation with radiological findings.**

SUMMARY

In this prospective study over a period of six months, in patients with AIDS and clinical CNS symptoms at KNH between July 1st 1998- December 31 1998, a total of 107 patients with CNS symptoms had CT scan examination, 4 of these patients had CT myelograms and the rest 103 had CT scan of the head examinations. The patients were grouped into six clinical groups depending on their clinical presentations. Various radiological reports were given.

Of the 107 patients, normal CT scan reports were given on 19 patients (17.8%), while the rest 88 patients (82.2%) had various abnormal radiological reports.

The sensitivity of CT on picking radiological abnormality in the study was (82.2%) while normal reports were given in (17.8%), this could be because the patient had normal scan, the cause of the neurological CNS abnormality could have been metabolic or failure of CT to pick very early and small cortical white matter lesions.

The commonest clinical group scanned was group 5 which presented with focal neurological signs and any other symptoms, it had a frequency of 43 patients (40.2%). This was followed by group 2 presenting with diffuse cerebral dysfunction; personality change; altered affect; poor memory; decreased concentration; confusion; ataxia alone or in combination this group had a frequency of 19 (17.8%), group 3 had 15 patients (14%) and it presented with headache, fever, photophobia and neck stiffness. Group 4 was the 4th commonest and it presented with fits 14 patients were registered in this group accounting for (13.1%), group 6 with 11 patients (10.3%) presented with depressed level of consciousness, fever plus confusion without meningism; fever plus headache without meningism lastly group 1 had the least number of patients with 5 (4.7%) and the clinical presentation was headache alone.

The commonest radiological finding was abscess in 32 patients (29.9%) followed by atrophy in 30 patients (28%), normal in 19 patients (17.8%), infarcts 15 (14%) encephalitis 10 (9.3%) hydrocephalus 5 (4.7%). Other radiological abnormalities which included myelopathy, Progressive Multifocal Leucoencephalopathy, bleed, sinusitis and mastoiditis were reported in 6 patients 5.6%).

After the analysis of the clinical presentation all the 107 patients had good reason for CT examination since positive radiological findings were found in all the six groups of clinical presentation and the pick up rate was high 82.2%.

STUDY JUSTIFICATION

With rapid increase in the number of patients who are infected with HIV/AIDS, it is important to study the radiological central nervous system (CNS) manifestations. It has been established elsewhere that about 10% of patients with HIV infection present with CNS symptoms as the presenting complaint, while about 30-39% of patients develop CNS complications later on as the disease progresses (1). No similar study has however been carried out in our set up therefore necessitating my current study.

It is important to neuroimage HIV patients with CNS complications/manifestations in order to diagnose patients with AIDS dimentia complex which is mainly indicated by brain atrophy, and to rule out other manageable conditions complicating the disease e.g. infections presenting as an abscess, meningitis or tuberculoma. Some CNS tumours e.g. lymphoma can also be suggested after CT scan. Early diagnosis of these manageable complication help the clinician in the management of patients which will also help in prolonging the life span of the HIV patients. Chest finding in HIV patients in our set up has been studied by Dr Ruiru in 1991.

Neuroimaging of these patients can be done using CT scan or MRI. No large study has been carried out to compare the two imaging findings in HIV patients. However several small studies have suggested that MRI is more sensitive than CT in detecting intracranial lesions in AIDS (2,3,4). MRI is more sensitive in detecting white matter abnormalities conversely lepto meningeal abnormalities are better detected with contrast enhancement CT scan examination (5). Therefore MRI and CT complement each other in the study of AIDS patients (3). If MRI and CT are equally available, it is reasonable to screen patients with MRI unless clinical symptoms clearly suggest meningitis. But if MRI is not readily available, CT scanning is a reasonable alternative (5). In our set up MRI is not available therefore I opted for CT scan study.

STUDY POPULATION

Inclusion Criteria: My study population included confirmed HIV positive patients with neurological symptoms, whom CT scan of the head examinations had been requested by the referring clinician in Kenyatta national Hospital.

Exclusion Criteria: (I). CT examination on sero negative patients or patient whose serological status is undetermined.

(ii) Head injury patients even if patient is seropositive.

TERMINOLOGIES

Hypodense, isodense and hyperdense means that the aternuation values of a certain measured area is lower, same or higher than the surrounding brain parenchyma respectively.

Pre and post contrast means before and after contrast medium introduction respectively. CNS - Central Nervous System,

CDC – Centre of Disease Control

MRI – Magnetic Resonance Imagining

CT – Computed Tomography Scanning

HIV – Human Immunodeficiency Virus

AIDS – Acquired Immunodeficiency Syndrome

SPECT – Single Photon emission computed tomography

HISTORY OF CT SCANNER

With introduction of CT scanner in 1972 by Godfrey Hounsfield as one of the medical imaging modalities, the diagnosis and clinical management of neurological disorders underwent a major revolution. CT scanning is one of the major developments in medical imaging in the 20th century. When imaging CNS however, MRI has been found to be superior to CT scan when dealing with posterior fossa and white matter lesions. There has been rapid technological advancement of the CT scanner from the first generation CT scanner which used to have a scan of 5 minutes to generate one crossectional image, to the newer scanners which use a very short scanning time. CT development earned Mr Godfrey Hounsfield a nobel price in year 1979.

A CT scanner consists of 3 major components:-

1. A high duty X-ray tube which produce X ray photons in range of 120KVP
2. An array of detectors which record the beams emerging from the patient.
3. A computer which analyses a large mathematical calculation of multiple X-ray attenuations. Depending on the matrix being used which can range from 256x256 to 512x512 , each numerical value is presented by a single picture element known as (pixel).

In CT scanning the cross-sectional anatomy of the slice is displayed on the screen.

When studying the head, routinely a 10 mm thickness slice is usually done. But when dealing with a small lesion or when studying the posterior cranial fossa a (1-2)mm thickness slice is done.

In generation of a cross-sectional image well collimated X-ray beam passes through the patient. In the patient there is partial absorption of the beam and the remaining photons which are not absorbed are transmitted through the patient and fall on the detectors. The detectors quantifies the photons and records digitally. This information is fed into a computer which gets different readings as the X-ray tube goes round the patient. The computer then analyses the data and a cross-sectional image is produced. The information of CT scan is stored in a computer disc as raw data. But since raw data occupies a lot of space on the disc, the data is usually erased after CT scan pictorial image has been produced. But if the raw data is required it can be protected.

CT scanning has an advantage over conventional radiography in that it has a high sensitivity to small differences in X-ray attenuation. Contrast resolution which is the ability to distinguish adjacent areas of differing radiodensity is superior in CT scanning as compared to conventional radiology. Utilization of high contrast resolution in CT scanning has rendered it very important in identifying minute intradermal pathology. In areas where a radiologist is not sure about the difference in attenuation, the utilization of the gray scale is employed which uses Hounsfield unit that ranges from +1000 to −1000. Positive numbers are given to structures such as bone and soft tissue which have a higher number than distilled or pure water which is assigned O; the negative numbers are assigned to fat and air. Since human eye can only distinguish a small number of shades of gray simultaneously, a system of windowing in CT scanning is employed, where the CT scan range number is spread to cover a range of full gray scale available on display system. The window level and width are manipulated to provide optimal viewing conditions e.g. when dealing with brain parenchymal pathology, soft tissue window is used and when dealing with bone pathology bone window is utilized.

Contrast medium administration in CT scanning is used to enhance the differences between lesions and surrounding parenchyma, to demonstrate vascular anatomy and vessel patency and to characterize lesions by their patterns of contrast enhancement. Defects in blood brain barrier associated with tumours, stroke, infections and other lesions allow contrast accumulation within abnormal tissue improving its visibility.

It is important to have knowledge of the patient history, physical examination findings and results of any investigations done, since this will help in the planning for the CT scan study.

The Kenyatta National Hospital CT scanner was installed in 1993 and it is a 3rd generation type of scanner.

LITERATURE REVIEW

Introduction

Aids is a terminal complication of long standing HIV infection affecting all systems of the body. It was first reported in 1981 to the CDC in United States, among young previously healthy, homosexuals and drug abusers who presented mainly with pneumocystis carini pneumonia, kaposis sarcoma and other opportunistic infections (6-9). Other presentation included mucosal candidiasis, chronic perianal ulcerative herpes simplex lesions. Later on Aids patients were reported in other groups of people e.g. haemophiliacs, who used blood donated by a large group of donors or drug users who shared needles with those who were infected with HIV (10). and this increased their exposure to HIV infections. Currently the major mode of transmission is by heterosexual method. From 1981 when the first case of Aids was reported until now there has been a rapid increase in the number of people infected with the virus. In 1996 it was estimated that 24 million people were infected with the HIV virus and it is estimated that by the year 2000 about 40 million people will be infected, majority of whom will be in Sub-Saharan countries.

Diagnosis

The virus that causes Aids was isolated in 1983 as the 3rd human retrovirus in lymphocytes of Aids victims (11-13).

1. While the assay of antibodies became widely available in 1985 by using Elisa method as a diagnostic screening test (14-16). Other methods are also available for testing for HIV antibodies and antigens, these include.
2. Western blot which is done according to standard modified method. (17-19)
3. Indirect immunoflourensence assay (IFA) used for screening or to substitute Western blot as a confirmatory test.
4. Radio Immunoprecipitation Assay (RIDA), is favoured as a confirmatory immuno assay though its use is restricted mainly in laboratories that have the facility and expertise to propagate HIV in continuous cell culture. The infected cells are grown in the radiolabled amino acid e.g. (35) S- Methionine

and S- cystine, so that the radio label can be incorporated into HIV-protein (20).

5 Rapid latex agglutination test is also used. (21-23). This has an advantage in that it is less costly and requires less skills. This utilizes a recombinant protein derived from a highly conserved region of HIV genone that is chemically linked to polystyrene.

6. P 24 antigen capture assay (23-25) can also be used and mainly after the serological method has been used. This is important in the management of the patient since it provides a quantitative measure of the level of viral P24 antigen present within serum or other body fluid, and therefore can serve as prognostic marker of disease activity over time (24). It is an important marker during the viraemia stage when there is rapid multiplication of the virus and the individual has not seroconverted. After seroconversion, the P24 antigen becomes bound to the antibodies and therefore the free antigen becomes less and at this stage the P24 antigen capture assay is less sensitive than the serological methods. Free P24 antigen decrease by about 3 fold during seroconversion.

Although HIV infection became widely evident in the early 80's, it is thought that the disease could have been existing in Central Africa before then since examination of stored serum collected from Africans at earlier periods between 1965-1975 was found to react with HIV I antibodies (8).

Transmission and Pathophysiology

The main modes of transmission are:-

1. Sexual contact that is both heterosexual and homosexual
2. Inoculation with contaminated blood and blood products
3. Vertical transmission i.e. from mother to child in pregnancy, during birth and on breastfeeding.

Once the virus is inoculated into the host it infect the cells which have CD4 (26-29) urface glycoprotein and it replicates by incorporating it's DNA genome into the host cell)NA By using it's gp 120 it selects the cells that have CD4 surface glycoprotein which nvolve mainly T-lymphocytes, (26) and Macrophages/Monocytes (31-32).

Marrow cells (34), astroglio, Oligodendroglio (35-36), epithelial langerhan cells (37), ollicular dendritic cells (38), brain endothelia cells, (35) and undefined cells of retina (30) re also infected. There is also selective and progressive depression of CD4-Lymphocytes in atients with Aids, showing a specific tropism of the virus to the cell bearing CD4 antigen.

In the CNS, macrophages within the brain tissue are the main target cells though ther cells may also be infected eg gliocells and vascular endothelial cells. Detection and ecovery of the virus in brain tissue strongly suggests that the virus may have a causal role in neurological abnormalities though the mechanism is not well known. It is thought that direct nfection of the brain cells and the replication of the virus may cause direct cytotoxicity to the rain cells, or alternatively the presence of viral antigen on the surface of the uninfected brain ells may make then target for cytotoxic effect by T-cells. Although macrophages and microglia cells as well as the multinuclear cells derived from these two cell forms are the principle target cells of the virus (39), speculation has now centered on indirect mechanism whereby, either the viral gene products released from infected cells or cytokines either eleased from infected cells or stimulated by the oligodendrocytes or astrocytes (40,41). The verlying impression engendered by these studies is that there is a significant involvement of NS by the AIDS virus that may produce neurological symptoms irrespective of the resence of opportunistic infections or tumours.

Patients with AIDS have a profound defect in T-cell lymphocyte function, haracterised by an absolute lymphopenia selective for T4 cells subset of T helper – inducer lymphocytes. The depletion of these lymphocytes as well as qualitative defect in T-cell function prevent a normal response to antigen material. There is also decreased antibody production. Therefore in addition to the potential direct effect of the HIV agents, the patient is susceptible to opportunistic infections and neoplasms.

Clinical presentation and opportunistic disease manifestation

The patients present with CNS symptoms which include headaches, photophobia, impaired concentration, forgetfulness, mental slowing, fits, unsteady gaits, leg weakness, loss of coordination, impaired handwriting and temors. They can also be apathetic, withdrawn, agitated, confused, changed in personality and hallucinating. In later stages of the illness patients may have delayed verbal response to absolute mutism, unaware of their illness and disorientation. On examination they may have weakness of both upper and lower limb, ataxia, double incontinence and paraplegia.

CNS manifestations may be accompanied by other symptoms of the disease in other organs e.g. diarrhoea and vomiting, abdominal pains, generalized lymphadenopathy and respiratory manifestations which include chronic cough, chest pain, dysponea, weight loss and night sweats. Cutaneous manifestation which include skin infections and other non infectious manifestation e.g. kaposis sarcoma, psoriasis and seborrheic dermatitis.

AIDs patients are more prone to opportunistic infections than the general population and this is because of their decreased immunity. The infective organisms include virus, bacteria, fungi and protozoa.

Opportunistic viral infections include cytomegalovirus (CMV) 42, whose inclusion bodies have been identified in patients brain tissue and it is thought to cause severe encephalitis. On CT scan or MRI of the brain the inclusion bodies are depicted as ventricular epencymitis or small cortical lesions which might be associated with brain atrophy.

Progressive multifocal leukoencephalopathy (PML) is another viral infection which produces white matter disease in AIDS. The incidence of PML in AIDS range from (2-7%) (43). The disease is caused by papova virus (J-C virus). Pathologically there is demyelination and necrosis of the white matter. Radiologically the abnormalities are seen in the white matter with no associated mass effect. On CT, focal zones of decreased density in the cerebral white matter that may be single or multiple are seen. These lesions progress with time to involve enlarging areas of white matter. Any portion of the cerebral hemispheres may be involved with slight parietoccipital prepoderance. There is usually no mass affect or contrast enhancement. CT usually underestimates the degree of impairment (44). MRI usually reveals areas of increased signal intensity in T2 weighted images in the subcortical white matter and centrum semiovale. There may be multiple areas of abnormality that are mainly asymmetrical and MRI usually shows more lesions than CT scan.

Other viral infections include herpes simplex (HSV 1 and II) and varicella zoster (VZV) virus. HSV causes mainly encephalitis. In the west herpes simplex infection is a common cause of fatal encephalitis in AIDS patients (45). The acute disease may present with confusion, disorientation and bizzare behaviour suggesting an acute dimenting illness. Both primary and recurrent infections can give rise to encephalitis, primary infections probably account for one-third of cases and most of these are under 18 years of age. The route of infection to the CNS is disputed but both olfactory tracts and trigeminal ganglia are suspected to be routes of infections.

The clinical presentation in the immunocompetent reflects the focal nature of the parenchymatous infection. Thus hemiparesis, dysphagsia, visual field loss or cranial nerve pulses are recognized presentations. CT and MRI may reveal oedema or necrosis of the temporal lobe. But detection of virus in CSF or biopsy remains the best way of making the diagnosis.

Varicella zoster virus (VZV) may cause CNS disease in three forms (46). It may cause multifocal encephalitis affecting primarily the white matter. Secondly it may cause vasculitis as in the setting of opthalmic zoster that can lead to infarction. Finally it can affect the spinal cord leading to myelopathy. About 10% of patients with HIV-1 infections will develop herpes zoster radiculitis representing reactivation of latent H zoster infection.(45) The white matter lesions are more effectively demonstrated with T2 weighted MRI than CT scan.

Fungal infections are known to cause neurological disorders in AIDS patients. About 10% of AIDS patients develop cryptococal meningitis with an early motality that approaches 15% (45) even with treatment, survival data compiled by the CDC for 3022 patients with extrapulmonary cryptococcosis (81% with menigitis) showed a median survival of 8.4 months. It is the commonest fungal infection of the CNS in AIDS patients (47). The causative organism is cryptococcus neoformans. It is a encapsulated solid yeast present in soil and especially prevalent in pigeon excreta. Virtually all cryptococcal diseases is caused by serotype A and D which occur in normal hosts and are more common in tropical and subtropical areas. It gains access to the body through the respiratory tract, though the pulmonary infection is usually asymptomatic. The blood borne spread can then affect any organ but CNS is commonly targeted.

In the CNS it causes a granulomatous meningitis sometimes associated with small cerebral cyst in the Virchow Robin space or granulomatous cryptococcomas. In immunocompetent hosts, it produces an indolent dimenting syndrome. Cryptococcus meningitis rarely develop when the CD_4 count is above 200. Clinically it may manifest as a pyrexia of unknown origin (PUO) but the usual presentation is of an ill, febrile patient with recent onset of headache who may be photophobic and confused. The history is from a few days to about 3 weeks in most cases. Diagnosis is made by CSF analysis which demonstrates the organism or its capsular polysaccharide. Lumbar puncture should be done promptly in patients suspected of cryptococcal meningitis because response to treatment depends on how early it is started.

Contrast enhanced CT scanning may demonstrate meningeal enhancement and small high signal (on T2 weighted MRI) are believed to correspond to virshow Robin spaces. Rarely, large non-enhancing masses are seen which are cryptococcomas. They are typically in basal ganglia. Hydrocephalus may follow obstruction of CFS by large cryptococcomas.

Other fungal infections which include coccidiodomycosis, histoplasmosis, blastomycosis, nocardia asteroides, candida and aspergillus have been reported in AIDS patients. CNS radiological findings may include leptomeningeal enhancement, focal masses which may or may not enhance post contrast scanning, focal cerebritis or hydrocephalus secondary to obstruction by fungal mass. Aspergillus though uncommon has a predilection to invade blood vessel wall and cause thrombosis, infarction and haemorrhage.

Bacterial infections are also common in AIDS patients. Among the opportunistic bacterial infections, syphilis and tuberculosis are on the rise. The use of illicit drugs, prostitution or contact with a prostitute have all been associated with rising syphilis incidence (CDC 1988b). The role of sexually transmitted diseases (STDS) as a co factor for HIV transmission is strongest for the diseases that cause genital ulceration; syphilis, herpes and chancroid. It has been suggested that the course of manifestation of neurosyphilis might be altered in the presence of immunodeficiency. The clinical spectrum of AIDS – related neurosyphilis appears to be no different from that in HIV negative patients, although it has been suggested that the course may be accelerated in the setting of cellular immunodeficiency. Thus the interval between infection and neurological complications may be shorter than usual. The clinical features of syphilis are divided into four stages:- primary, secondary, latent and tertiary. In primary syphilis lesions develop at the site of inoculation,

with an incubation period averaging 21 days. The chancre is a single, rounded, painless lesion with raised borders and rubbery or hard consistency.

In the secondary stage of syphilis, there is haematogenous dissemination with fever, malaise and generalized lymphadenopathy. Tertiary syphilis develops in one third of untreated patients and it includes gummatous disease and cardiovascular involvement with aortitis.

Neurosyphilis tends to develop during secondary or tertiary stages. Neurosyphilis is divided into asymptomatic and symptomatic and can affect the CNS at different levels, e.g. eyes, meninges, brain parenchyma and spinal cord.

In asymptomatic patients there are no neurological symptoms or signs but CSF is positive for VDRL. In the spinal cord syphilis can cause syphilitic meningomyelitis, pachymeningitis, spinal cord gumma, spinal vascular syphilis, tabes dorsalis and syringomyelia. On CT scan, features of leptomeningeal enhancement in case of syphilitic meningitis are seen. Infarction and gummatous lesions can also be noted. The gummatous disease of the CNS which may involve brain or spinal cord is an unusual manifestation of neurosyphilis in the HIV infected individuals. When they occur they may be confused with other more common intracranial mass lesions such as Toxoplasmosis. (48)

Tuberculosis (TB) is a very important infection as far as the AIDS pandemic is concerned. TB involving the CNS is now recognized as a common complication of AIDS. The AIDS pandemic has led to an increase of TB in countries such as USA where it had been on the decline, and it is particularly common in Africa. It has been suggested that HIV-positive patients should be skin tested and those whose response is positive (>5mm induration) should receive prophylaxis with isoniazid. Those whose response is negative (<5mm induration) should not receive BCG vaccination because of the risk with a live vaccine in immunocompromised individuals. About 30-40% of TB patients are seropositive for HIV in New York, with a wide variation elsewhere depending on town and race(45). Rising incidence of TB associated with AIDS epidemic has been reported from a number of African states.

The neurological complications of TB in CNS include TB meningitis, direct effect of a granuloma in the brain parenchyma or an endarteritis with ischaemic foci. Patients with TB meningitis just like any other meningitis presents with malaise, fatigue, headache, neckstiffness, fever, confusion and in severe cases patients might present in coma.

CT scanning with contrast reveals meningeal enhacement in cases of TB meningitis. Other CT findings include focal lesions and hydrocephalus secondary to obstruction, ring enhancing lesions also occur in case of abscess formation. Shafer(49) in his study of 52 HIV infected patients with no evidence of clinical CNS TB, head scans of all the 52 patients revealed that four of them had enhancing brain lesions which responded to anti TB treatment. This suggest that HIV infected patients who develop TB at any site should probably have cranial CT scan or MRI done. It also suggested that ring enhancing lesions may be due to TB if they are not responding to anti toxoplasma treatment or if the patient has pulmonary TB. Tuberculous lesions in the brain may be large enough to behave as space occupying lesions. Vertebral and spinal cord abscess has also been described. The diagnosis depends on a high index of suspicion, the chest radiograph and the CSF findings of a reduced glucose levels. In presence of active pulmonary TB infection, and a negative toxoplasma antibody test, focal neurological and/or brain imaging findings would be sufficiently suspicious of CNS Tuberculosis.

Other rare opportunistic bacterial infections encountered in the CNS in AIDS patients include mycobacterium avium intracellulare and listeria monocytogenes. They may cause encephalitis, abscess or focal lesions and meningitis.

Among the parasitic opportunist infections, Toxoplasmosis is the most commonly encountered infective organism in AIDS patients. Prior to 1980 cerebral Toxoplasmosis was a rare infection encountered in immunosuppressed patients e.g. renal transplant recipients and patients with cancer and those on cancer chemotherapy. With AIDS pandemic it has become a frequent cause of encephalitis and abscess. CNS infection with Toxoplasma gondii which is an obligate intracellular protozoa causes necrotic abscesses that are often multifocal and scattered throughout the cerebral hemispheres with a predilection for basal ganglia. The oocysts are excreted in the faeces of cats, the definitive hosts. The ingestion of which lead to dissemination of infection to a new host. It then may manifest as a fever like illness with lifelong encystment in any tissue. The T-helper lymphocytes and activated macrophages are important in containment and elimination of the parasite. With HIV infection which leads to failure of suppression of the development of the pathological infection at sites of encystment e.g. in the brain the infection flairs up. Toxoplasmosis is thought to occur in about a quarter of HIV infected patients (50). Clinically the patients present with fever, headache altered

mentation, seizures and focal neurological signs. If it is involving the cerebellum patients may present with ataxia and dysathria, neck stiffness may also occur.

The clinical features of Toxoplasmosis and primary CNS lymphoma may not be so distinct. On imaging the two conditions may demonstrate multiple contrast enhancing mass lesions. However areas of oedema usually surround toxoplasma lesions, producing mass effect leading to shift of the surrounding structures.

Toxoplasma abscesses are more typically small "ring" enhancement lesions whereas lymphoma are larger with more heterogenous enhancement. The abscesses show an unexplained predilection for the basal ganglia, but other lobes may also be involved. On imaging, MRI is more sensitive than computed tomography frequently showing lesions that are not identifiable even on contrast CT (51). When the lesion is small enhancement may be "solid" rather than the "ring" pattern.

It is important to note that the number of CT or MRI lesions and their radiological characteristics cannot reliably differentiate toxoplasmosis from lymphoma or any other cause of brain abscess. CSF assay for elevated proteins and pleocytosis plus serological testing for anti-toxoplasma IgG should be done (52). CSF is not always obtainable because the mass effect may be associated with raised intracranial pressure, and lumbar puncture is therefore deferred to avoid tonsilar herniation. The diagnosis in practice depends on suspicious clinical history and competent neuroimaging by CT or MRI.

Other rare documented parasites causing CNS infections in AIDS patients include acanthamoeba, trypanosoma and pneumocystis. Amoeba may cause meningo-encephalitis, brain abscess, necrotizing thromboangitis and extensive demyelination associated with amoebic trophozoites (53).

In AIDS in general, there is an increased incidence of some malignancies. These cancers appear to develop because of a chronic and profound defect in immune surveillance associated with HIV infection. Viruses and other co-factors, in addition to HIV, may induce malignant transformation directly or indirectly. AIDS patients are more prone to develop non Hodgkin's lymphoma and kaposis sarcoma. These two malignancies account for 95% of cancers in AIDS patients (45). Other cancers which are encountered in AIDS patients include Ca cervix, carcinoma of vulvae and squamous cell carcinoma of the skin.

Neurological involvement by kaposis sarcoma (KS) is relatively rare, but metastatic lesions have been described in the brain. They behave like any other brain metastasis, with

contrast enhancement at the white-gray matter junction (54). CNS KS lesions can bleed, producing coma or stroke-like illness.

Systemic non Hodgkin's lymphomas have been found to occur in AIDS patients 60 times more than in the general population (55). CNS lymphoma can either be primary or metastatic from other primary sites. The CDC data of up to 1989 has 548 cases of primary CNS lymphoma which represent an increased incidence of about 1000 fold compared to what is expected in the general population (45). The main cell type is a B. cell derived small cell, other cell types include large cell immunoblastic and rarely angiocentric T cell tumours. Patients present with headache, behavioral changes, seizures and focal deficits, memory loss and confusion. Differential diagnosis include PML, toxoplasmosis, abscess, infarct and haemorrhage.

Most tumours have multiple foci at autopsy and imaging reveals multiple lesions too. A large solitary lesion should suggest primary CNS lymphoma. On CT scan the lesions are depicted as low, or less commonly, as high density with mass effect and usually enhances. Some lesions may show some haemorrhagic component. In most cases the nature of enhancement is irregular or ring after contrast. Periventricular spread may also be seen. It is difficult radiologically to differentiate lymphoma from infective processes e.g. toxoplasmosis. Thallium 201 spect scanning can differentiate lymphoma from infective processes e.g abscess. The radioisotope is taken up by tumour cells and produces a hot spot while abscesses remain cold (56). Another adjuctive method for differential diagnosis of focal brain lesions in AIDS is MR spectroscopy. Current results indicates that Toxoplasmosis and Lymphoma have highly distinctive chemical features (57). In Toxoplasmosis lesions, lactate and lipids are elevated, with absence of all normal brain metabolites, whereas an increase in choline could be observed in Lymphoma.

A few CNS lymphomas may not enhance at all after contrast and on examination of CSF, tumour cells may or may not be revealed. So in such cases biopsy may be the only reliable way of making a diagnosis. If there is doubt about whether a lesion is infective or neoplastic therapeutic trial e g of anti toxoplasma treatment is given for 2 weeks. Lack of a clinical or radiological response will then show that a brain biopsy is indicated. The biopsy can be done by stereotactic CT or MRI guided procedure or for larger lesions requiring decompression, open craniotomy can be done.

Other CNS tumours e.g. cerebral gliomas, meningiomas, pituitary adenomas have been encountered in AIDS patients but there is no evidence to support increased incidence of these tumours. (45)

Cerebrovascular complications in AIDS patients are sufficiently frequent implying that it is in some way a complication of HIV infection or sequelae (58). The vascular lesion can either be infarctive or haemorragic. Ischaemic cerebrovascular lesions may result from embolism. This may follow cardiac disease which may be secondary to viral or toxoplasma myocarditis and congestive cardiomyopathy which may result from zidovudine toxicity.

Cerebral venous thrombosis associated with cachexia and dehydration can also occur. The AIDS patients may suffer vasculitic changes in intracerebral vessels which can be due to opportunistic infections e.g. candida albicans, aspergillus fumigatus, cryptococcus, cytomegalovirus, syphilis, herpes simplex and tuberculosis. HIV itself might induce vasuclitis too.

Anticardiolipin antibodies develop in some AIDS patients and may be the underlying cause of ischaemic cerebral infarction. Cerebral haemorrage may also occur as a result of thrombocytopenia which may occur in AIDS patients because of some factors which include:- depression of bone marrow progenitor cell, immunosupression by the virus and pancytopenia occurring in AIDS patients following marrow depression by the drugs used to manage the disease complications e.g. anti cancer drugs and anti viral drugs.

HIV infection is a multi-organ disease and a patient may present with neurological complications resulting from other organ failures. For example encephalopathy may result from hypoxia arising from severe pulmonary disease, uraemia from end stage renal failure, septicemia, disseminated intravascular coagulation and toxic effects of drugs administered. Electrolyte imbalance and dehydration occurring after prolonged diarrhoea and vomiting may lead to CNS manifestation. In the metabolic brain disorder CT scan and MRI depicts a normal finding, but the metabolic disorder may accelerate the development of AIDS dementia complex.

MATERIALS AND METHODS

Patients coming for CT scan examination in KNH X-ray department, usually have CT scan request form countersigned for the examination by either a consultant or a registrar in the department. Before signing, the request form is studied to make sure that the examination is justified and to ensure that the relevant clinical summary is available. After signing the patients are booked. But if a patient needs an emergency examination he is not booked but the examination is done as an emergency.

For the study the clinical summary and the serological status of the patients were obtained from the request form and the notes of the patients, which are usually available when the CT scan is being done.

CT scanning was performed on all patients using a 3rd generation CT scanner Philips Tomoscan CX1Q machine.

4 patients had CT myelogram examinations while the rest 103 patients had CT scan of head examinations. The patients were scanned in supine position. For the head examination continuous axial CT scans of 10mm cuts were done from the base of the skull to the vertex. Though when studying posterior fossa or a tiny lesion 2mm cuts were done. Contrast media was introduced in 67 of the patients and single dose immediate scans were done. Other set ups advocate for a double dose either immediate or delayed scanning. Double dose delayed scanning is reported to be superior in some lesions especially in the small white matter lesions.

By using the soft tissue and bone windows, the brain parenchyma, ventricular system, sulci, cisterns, bones, sinuses and soft tissues of the head were studied. The report was given by the consultants. A typed report, accompanied by printed CT scan images were given to the referring clinician to further manage the patient accordingly.

For data collection the necessary information which included the age, sex, unit number, clinical presentation, use of contrast media and the radiological finding were noted.

Ethical consideration

Patients names were not recorded in order to retain confidentiality. For referral purposes it is only the unit No., which was recorded. The CT scan was carried out only in those patients whose examination was requested. The request forms were countersigned by

the consultant radiologist or the registrar in the department of radiology, therefore ethical problems were not anticipated. However, the proposal was reviewed and approved by the ethical committee of KNH.

RESULTS

Results will be presented in forms of tables 1 to 10.

TABLE I: SEX DISTRIBUTION

SEX	FREQUENCY	PERCENT
Male	61	57.0
Female	45	42.1
Not indicated	1	0.9
TOTAL	107	100

Male:Female ratio 1 : 0.74

TABLE II: AGE GROUP DISTRIBUTION

AGE GROUP	FREQUENCY	PERCENT
Less or equal 10 years	7	6.5
11-20 years	3	2.8
21-30 years	23	21.5
31-40 years	39	36.4
41-50 Years	22	20.5
>50 years	1	0.9
Adult	9	8.4
Not indicated	3	2.8

The mean age was 33.43 years with a standard deviation of 12.1 years. Age ranged from 8 months to 52 years for those whose age was indicated.

TABLE III: DISTRIBUTION OF CLINICAL PRESENTATION

CLINICAL PRESENTATION	FREQUENCY	PERCENT
Headache alone (GP 1)	5	4.7
Diffuse cerebral dysfunction; personality change; altered affect; poor memory; decreased concentration; confusion; ataxia alone or in combination (GP 2)	19	17.8
Headache, fever, photophobia, neck stiffness GP 3)	15	14.0
Fits (GP 4)	14	13.1
Focal neurological signs and any other symptoms (GP 5)	43	40.2
Depressed level of consciousness; fever + confusion without meningism; fever + headache without meningism (GP 6)	11	10.3
TOTAL	107	100

TABLE VI: DISTRIBUTION OF CT FINDINGS

CT FINDINGS	FREQUENCY	PERCENT
Brain atrophy	17	15.9
Abscess	25	23.4
Encephalitis	5	4.7
Infarct	6	5.6
Normal findings	19	17.8
Mass /tumour	8	7.4
Meningitis	2	1.9
Others	5	2.8
Atrophy & abscess	2	4.7
Atrophy & encephalitis	3	0.9
Atrophy & infarct	5	0.9
Atrophy & hydrocephalus	1	2.8
Abscess & encephalitis	1	0.9
Abscess & hydrocephalus	3	2.8
Encephalitis & infarct	1	0.9
Infarct & others	1	0.9
Atrophy, abscess & meningitis	1	0.9
Atrophy, infarct & meningitis	1	0.9
Infarct, meningitis & hydrocephalus	1	0.9

TABLE IV: DISTRIBUTION OF INDIVIDUAL CT FINDINGS

CT SCAN FINDINGS	FREQUENCY	PERCENT
Brain atrophy	30	28.0
Abscess	32	29.9
Encephalitis	10	9.3
Infarct	15	14.0
Normal CT findings	19	17.8
Mass	8	7.5
Meningitis	5	4.7
Hydrocephalus	5	4.7
Others #	6	5.6

- Others include: myelopathy, leucoencephalopathy sinusitis and acute thalamic bleed.

TABLE V: DISTRIBUTION OF INDIVIDUAL CT FINDINGS BY CLINICAL PRESENTATION

CT FINDINGS		GP 1	GP 2	GP 3	GP 4	GP 5	GP 6
Brain atrophy	N	1	7	7	2	11	2
	%	3.3	23.3	23.3	6.7	36.7	6.7
Abscess	N	3	5	-	6	13	5
	%	9.4	15.6	-	18.8	40.6	15.6
Encephalitis	N	-	1	1	1	6	1
	%	-	10.0	10.0	10.0	60.0	10.0
Infarct	N	-	5	3	-	6	1
	%	-	33.3	20.0	-	40.0	6.7
Normal findings	N	1	3	3	4	6	2
	%	5.3	15.8	15.8	21.1	31.6	10.5
Mass/?tumour	N	-	1	1	1	4	1
	%	-	12.5	12.5	12.5	50.0	12.5
Meningitis	N	-	2	3	-	-	-
	%	-	40.0	60.0	-	-	-
Hydrocephalus	N	1	2	1	-	-	1
	%	20.0	40.0	20.0	-	-	20.0
Others	N	-	1	1	1	3	-
	%	-	16.7	16.7	16.7	50.0	-

N = Number ; % = percent

TABLE VII: DISTRIBUTION OF CT FINDINGS BY CLINICAL PRESENTATION

CT FINDINGS		GP 1	GP 2	GP 3	GP 4	GP 5	GP 6
Brain atrophy	N	1	3	2	1	9	1
	%	5.9	17.6	11.8	5.9	52.9	5.9
Abscess	N	2	3	1	5	11	3
	%	8.0	12.0	4.0	20.0	44.0	12.0
Encephalitis	N	-	-	-	1	3	1
	%	-	-	-	20.0	60.0	20.0
Infarct	N	-	2	-	-	3	1
	%	-	33.3	-	-	50.0	16.7
Normal findings	N	1	3	3	4	6	2
	%	5.3	15.8	15.8	21.1	31.6	10.5
Mass/tumour	N	-	1	1	1	4	1
	%	-	12.5	12.5	12.5	50.0	12.5
Meningitis	N	-	-	2	-	-	-
	%	-	-	100	-	-	-
Others	N	-	1	1	1	2	-
	%	-	20.0	20.0	20.0	40.0	-
Atrophy & abscess	N	-	-	-	1	-	1
	%	-	-	-	50.0	-	50.0
Atrophy & encephalitis	N	-	1	1	-	1	-
	%	-	33.3	33.3	-	33.3	-
Atrophy & infarct	N	-	2	2	-	1	-
	%	-	40.0	40.0	-	20.0	-
Atrophy & hydrocephalus	N	-	-	1	-	-	-
	%	-	-	100	-	-	-
Abscess & encephalitis	N	-	-	-	-	1	-
	%	-	-	-	-	100	-
Abscess & hydrocephalus	N	1	1	-	-	-	1
	%	33.3	33.3	-	-	-	33.3
Encephalitis & infarct	N	-	-	-	-	1	-
	%	-	-	-	-	100	-
Infarct & others	N	-	-	-	-	1	-
	%	-	-	-	-	100	-
Atrophy, abscess & meningitis	N	-	1	-	-	-	-
	%	-	100	-	-	-	-
Atrophy, infarct & meningitis	N	-	-	1	-	-	-
	%	-	-	100	-	-	-
Infarct, meningitis & Hydocephalus	N	-	1	-	-	-	-
	%	-	100	-	-	-	-

N = number; % = percent

TABLE VIII: DISTRIBUTION OF CT FINDINGS BY CONTRAST #

TABLE VIII: DISTRIBUTION OF CT FINDINGS BY CONTRAST #

CT FINDINGS	CONTRAST		NO CONTRAST	
	N	%	N	%
Brain atrophy	3	42.9	4	57.1
Abscess	26	100		
Encephalitis	3	100	-	-
Infarct	6	100	-	-
Normal findings	2	28.6	5	71.4
Mass/tumour	8	100		
Meningitis	2	100	-	-
Others	2	100	-	-
Atrophy & abscess	2	100	-	-
Atrophy & encephalitis	1	100	-	-
Atrophy & infarct	3	100		
Abscess & encephalitis	1	100	-	-
Abscess & hydrocephalus	2	100		
Encephalitis & infarct	1	100	-	-
Infarct & others	1	100	-	-
Atrophy, abscess & meningitis	1	100	- -	-
Atrophy, infarct & meningitis	1	100	-	-
Infarct, meningitis & hydrocephalus	1	100	-	-

Only 67 records had indication that contrast media was used.

TABLE IX: DISTRIBUTION OF TYPE OF LESION BY SITE

SITE	ABSCESS N %	INFARCT N %	MASS N %	PML N %	BLEED N %	CEREBRITIS N %
SINGLE: Basal ganglia	3 9.4	5 33.3	1 12.5			
Thalamic	1 3.1		1 12.5		1 100	
Parietal	3 9.4	2 13.3	3 37.5			
Sacrum			1 12.5			
Cerebella	2 6.3	2 13.3				
Caudate		1 6.7				
Frontal	1 3.1	2 13.3				
Occipital		1 6.7				
Temporal	2 6.3					
MUTIPLE: Occipital	1 3.1					1 100
Parietal – occipital	3 9.4		1 12.5			
Cerebella + parietal	1 3.1					
Frontal + thalamic	2 6.3					
Occipital, parietal + frontal	1 3.1					
Rt frontal, both frontal	1 3.1			1 100		
Cerebellum, parietal, caudate	1 3.1					
Parietal + caudate	1 3.1					
Frontal – parietal	1 3.1					
Frontal – temporal	1 3.1					
Basal ganglia		1 6.7				
Frontal, temporal + parietal	1 3.1					
Parietal-temporal	2 6.3					
Parietal, frontal, basal	1 3.1					
Cerebellum		1 6.7				
Temporal-thalamic	1 3.1					
Basal + temporal	1 3.1					
Cortical	1 3.1					
Rt caudate + thalamic		1 12.5				
TOTAL	32	15	8	1	1	1

TABLE X: DISTRIBUTION OF TYPE OF LESION BY TYPE OF SITE

TYPE OF SITE	ABSCESS	INFARCT	MASS	PML	BLEED	CEREBRITIS
Single	12 37.5	13 86.7	6 75.0	- -	1 100	- -
Multiple	20 62.5	2 13.3	2 25.0	1 100	- -	1 100

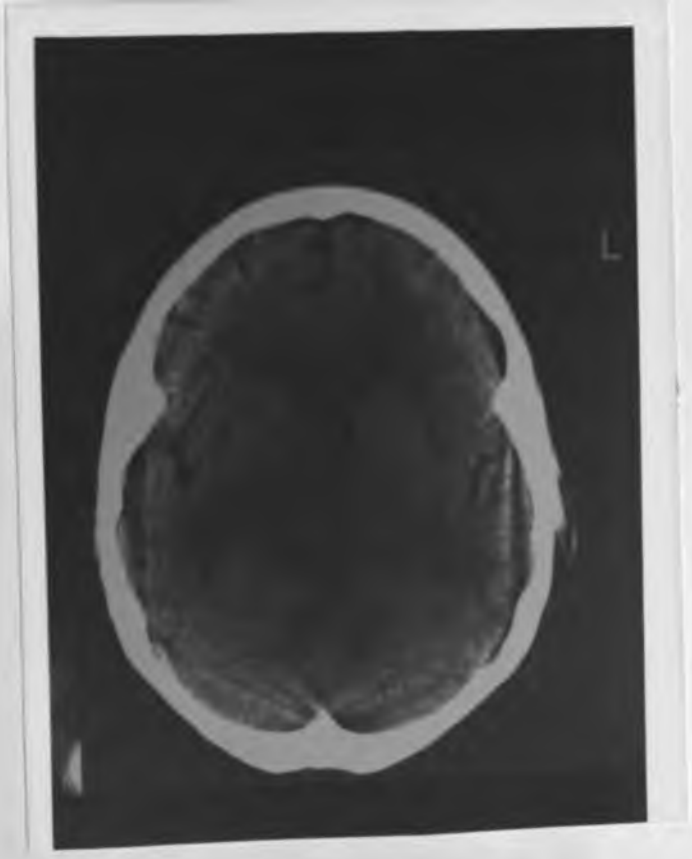
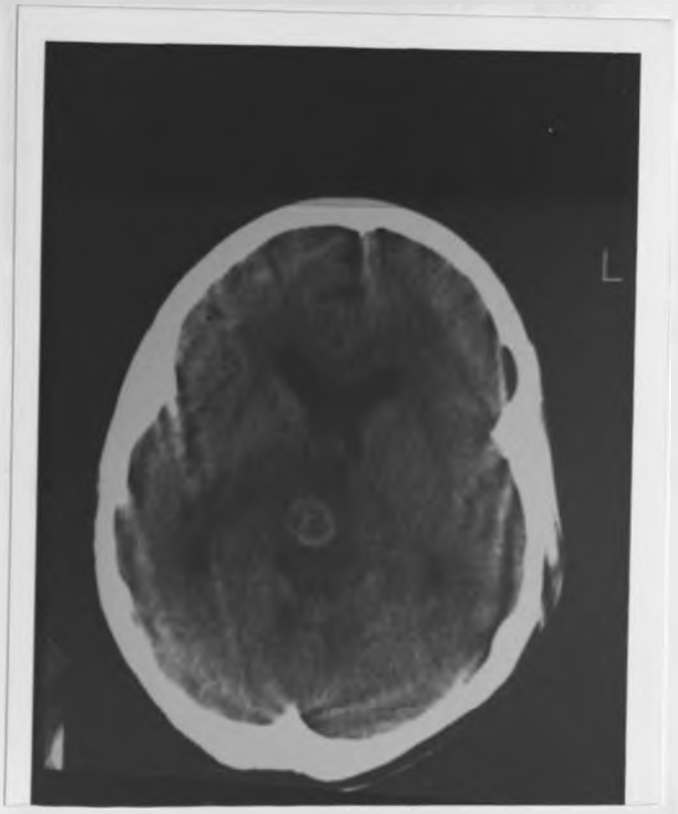


FIG 1 A **Shows a hypodense lesion in the region of right Thalamus.**



1 B **Same patient as in fig 1A showing ring enhancing lesion with surrounding oedema suggestive of an abscess.**

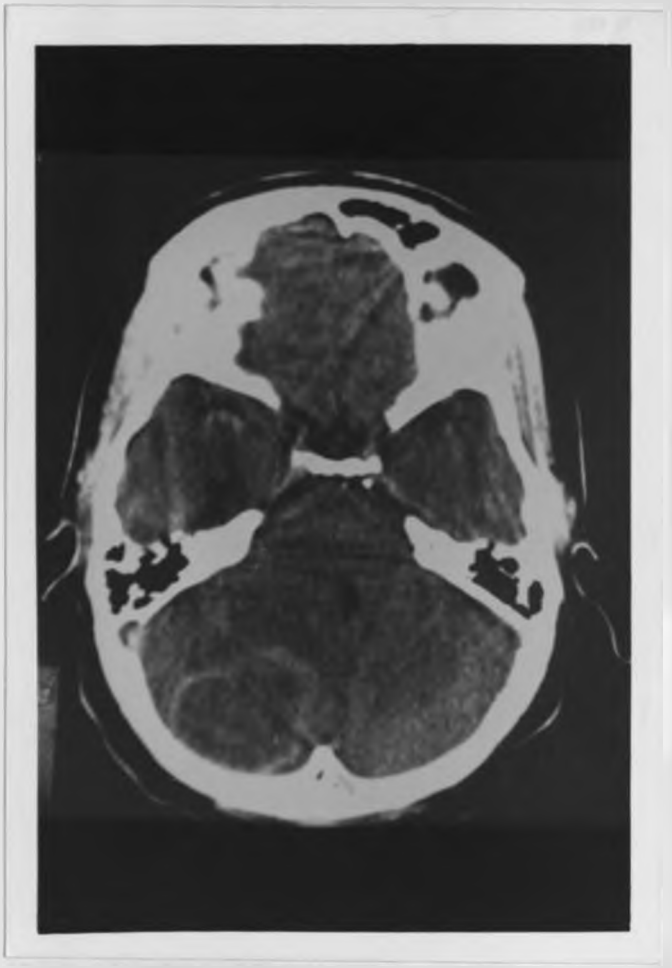


Fig 2 Shows an isodense ring enhancing lesion in the Rt cerebella posteriorly suggesting an abscess

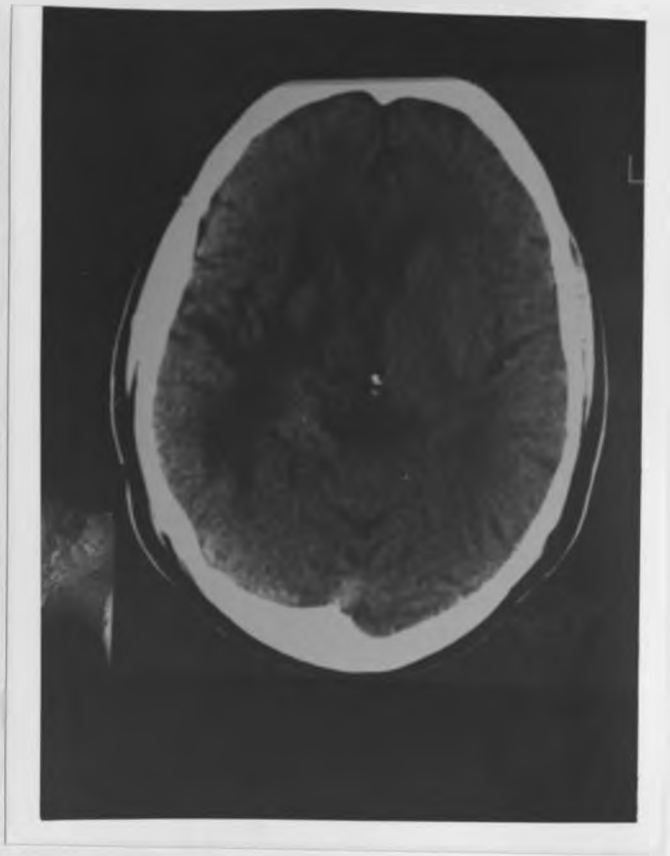
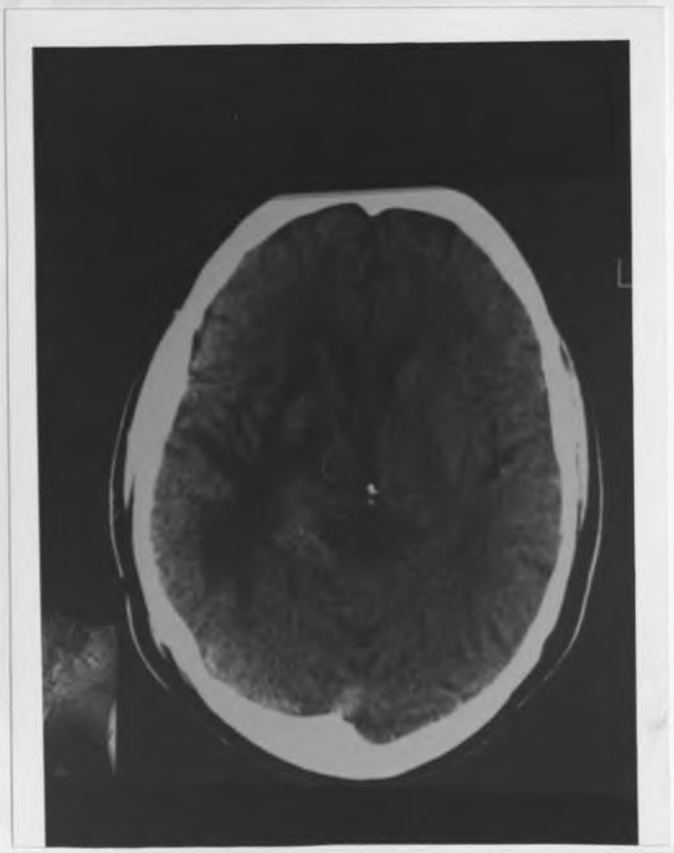


Fig 3 A



3B

Figs 3 (A&B) Pre and post IV contrast images showing multiple ring enhancing lesions on the right parietal and Rt thalamic region with associated oedema, features are suggestive of multiple abscesses.

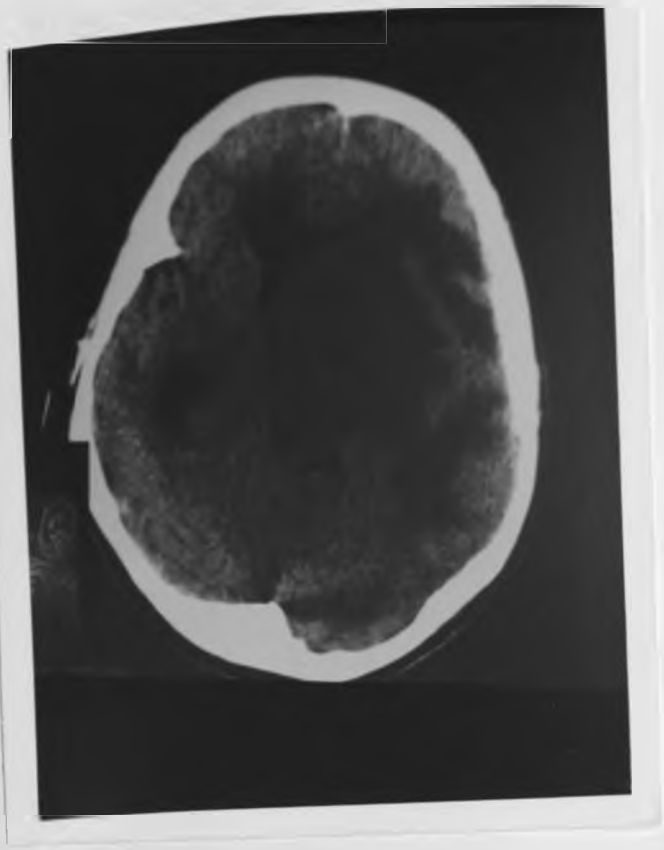


Fig 4A

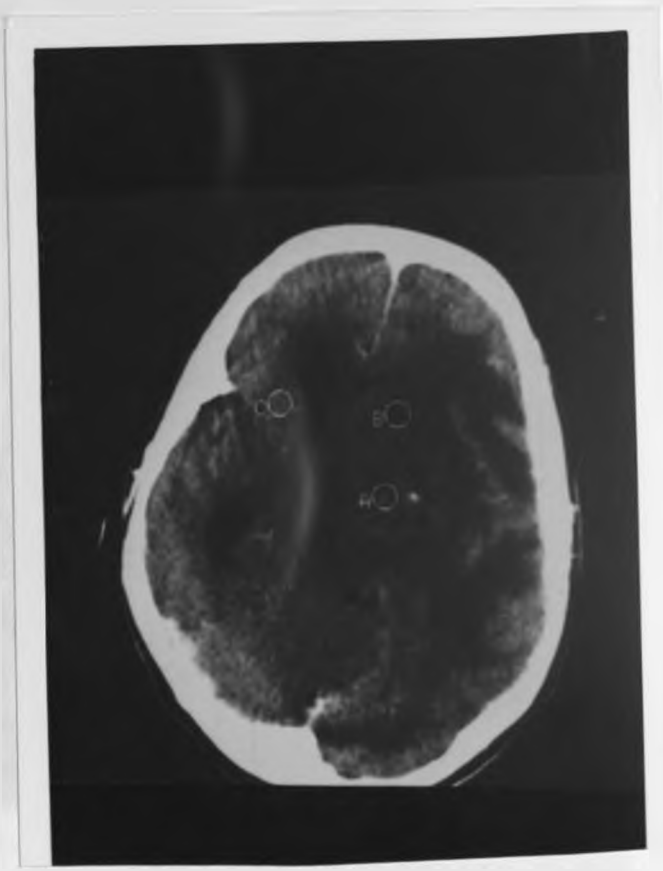


Fig 4B

Figs 4 (A & B)Pre and post IV contrast images showing an ill defined mass in the left basal ganglia, post contrast no significant enhancement noted. There is also associated compression of the ventricular system and shift of the midline to the right, features suggestive of a tumour.

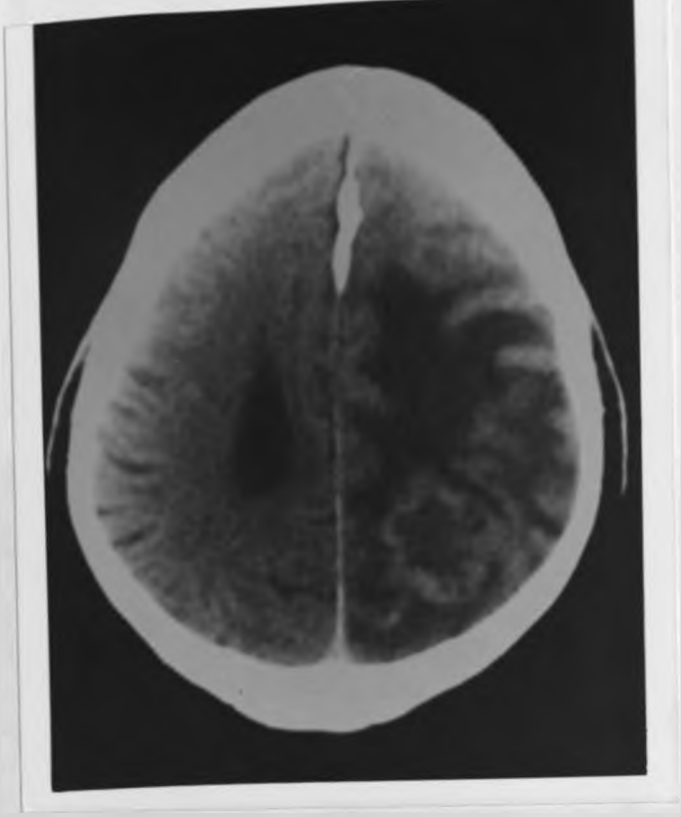


Fig 5A

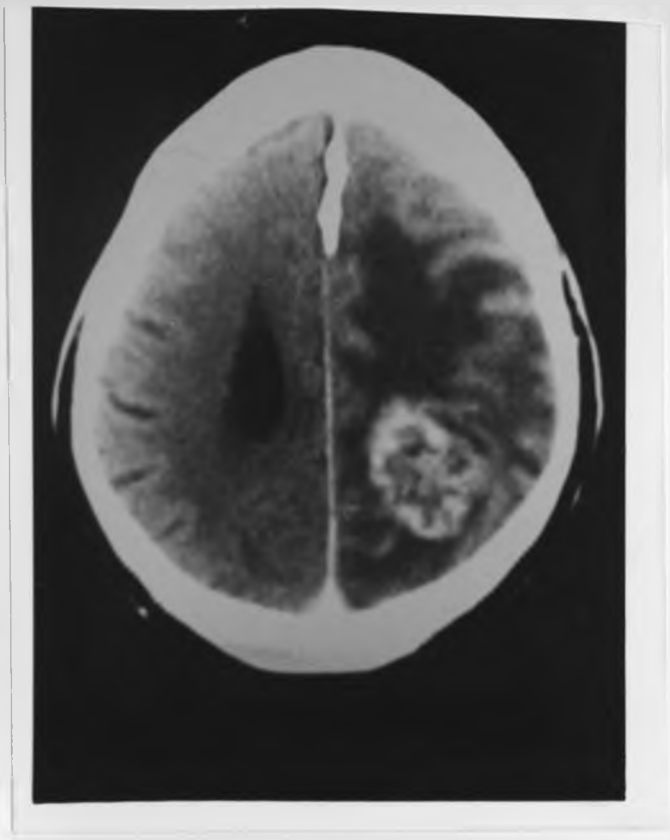


Fig 5B

Figs 5(A&B) Pre and post IV contrast images showing an irregular enhancing mass on the left parietal region with associated brain oedema suggestive of a tumour.

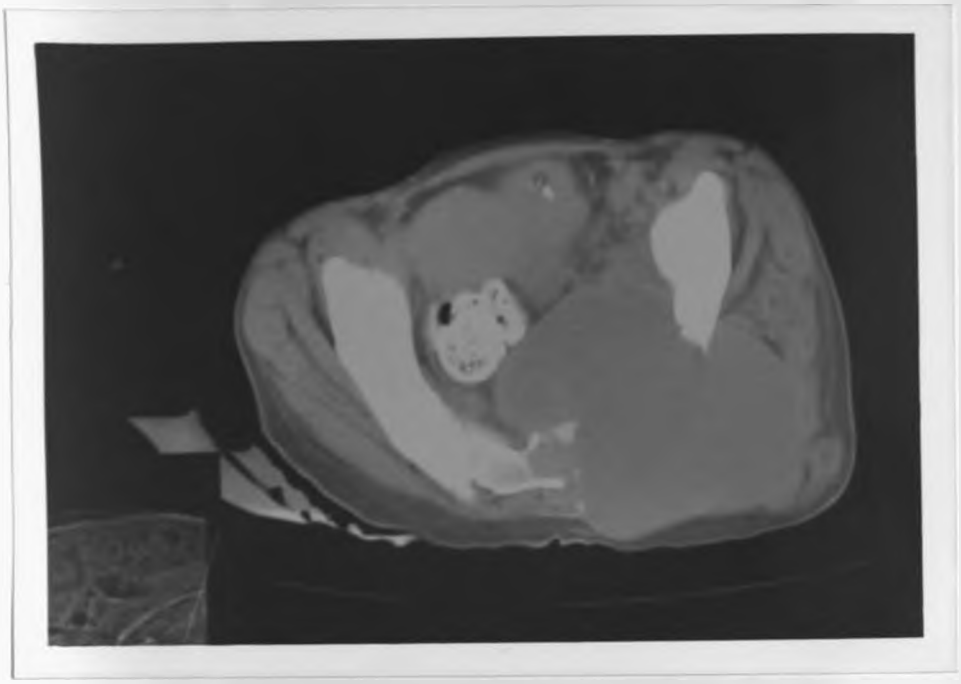
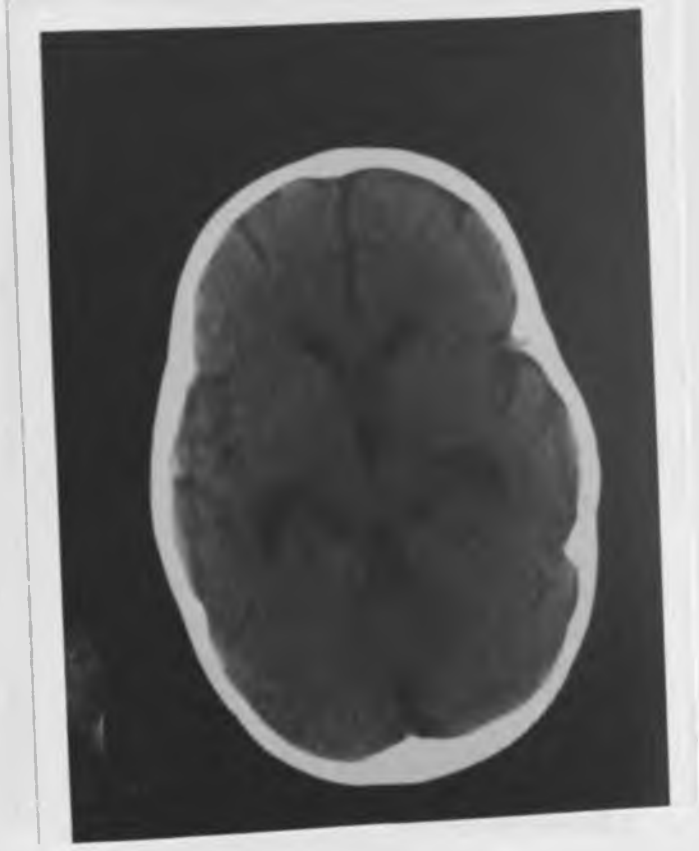


Fig 6 - CT Myelogram of the lower lumbar region showing a soft tissue mass with destructive effect to the left sacrum and ilium.



(7A)

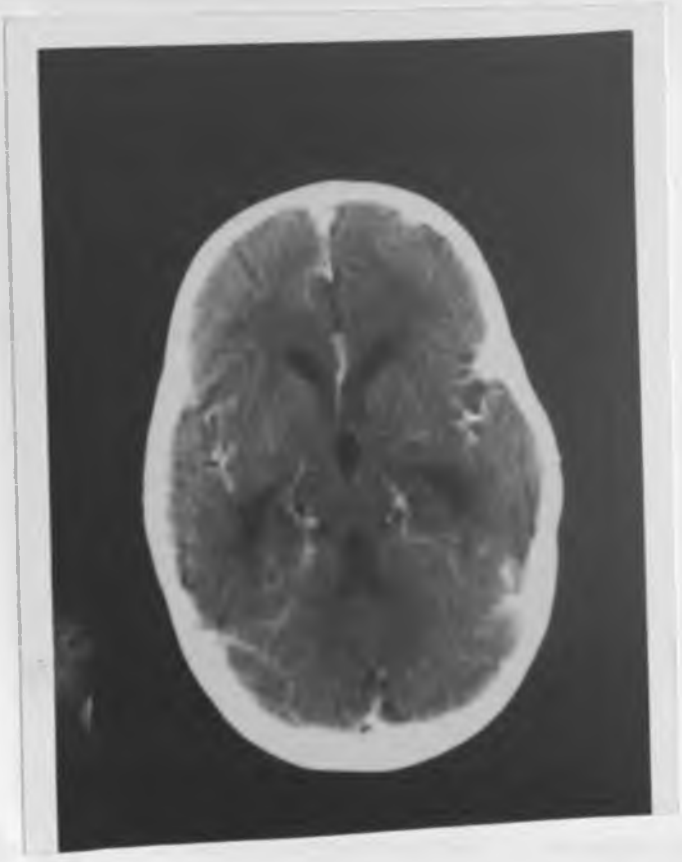


Fig 7B

Fig 7 (A&B) pre and post contrast images showing slight dilatation of the ventricular system, in post contrast leptomeningeal enhancement is noted suggesting meningitis.

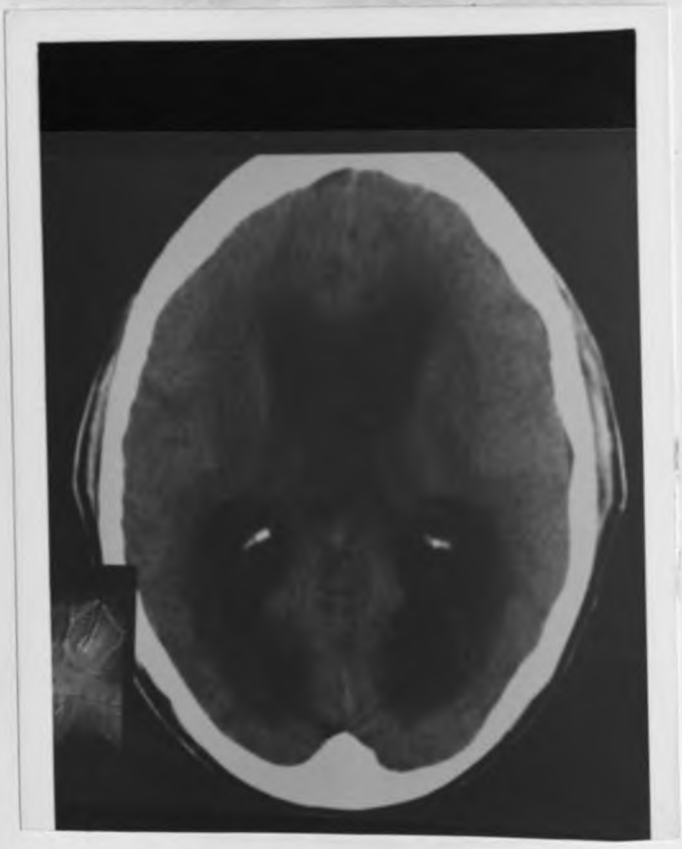


Fig 8A

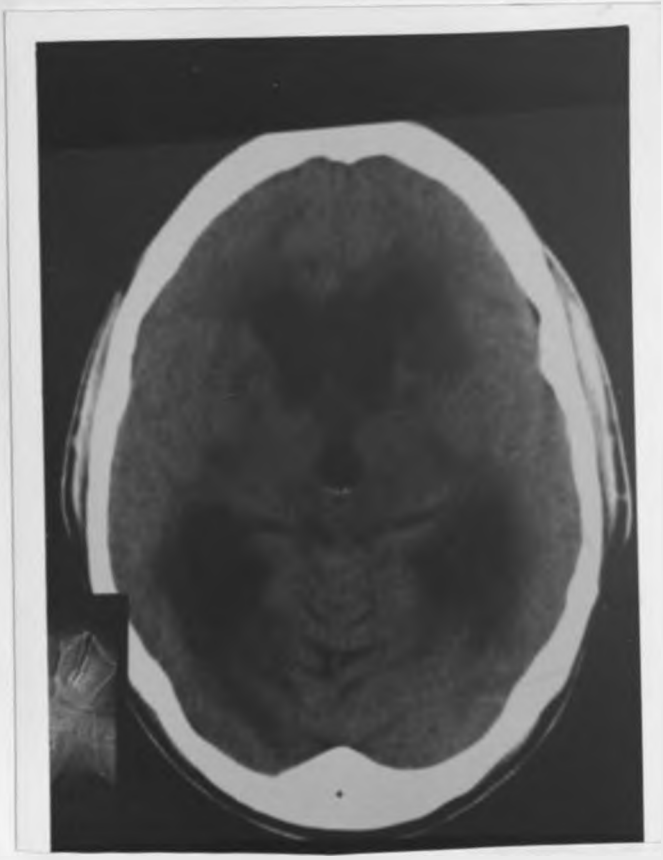
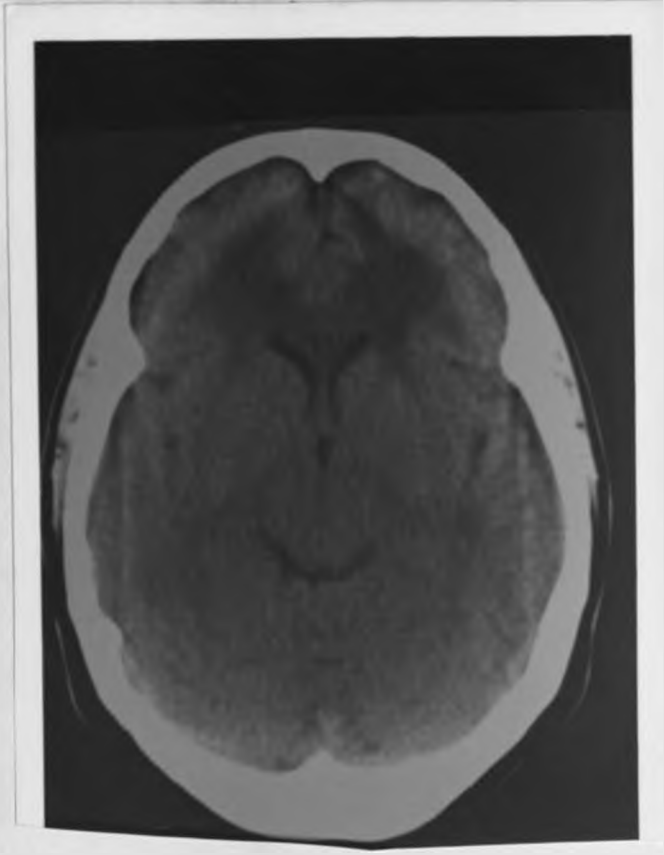


Fig 8B

Fig 8(A&B) Same patient, shows obstructive hydrocephalus at the aquaduct of Sylvia . A Hypodense lesion in the region of the left caudate neuclus which does not enhance post contrast is noted suggesting an infarct.



9A

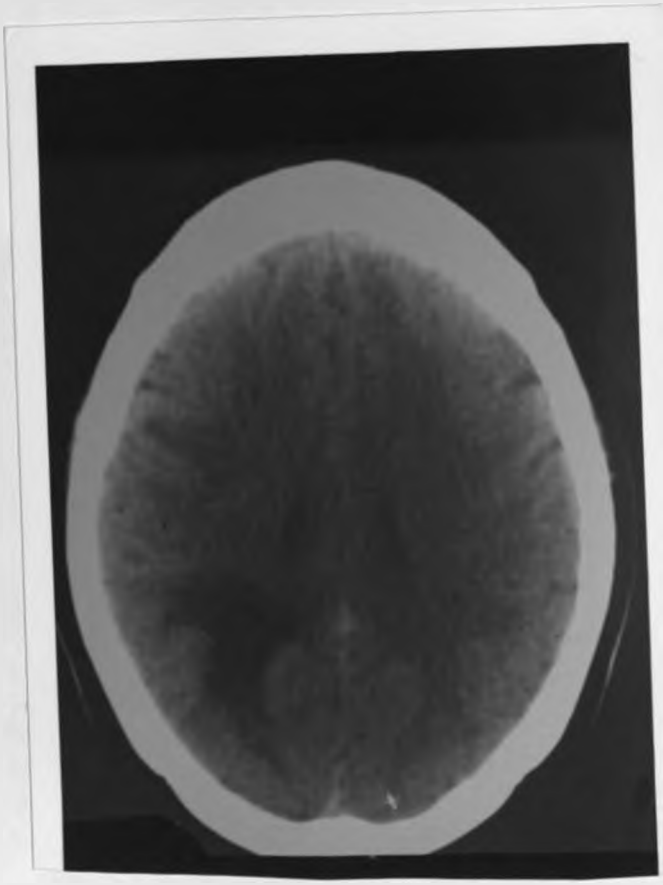


Fig 9B

Fig 9(A&B) Shows bilateral symmetrical hypodense lesion in the frontal cortical white matter, another hypodense lesion is noted in right parietal occipital white matter. Post contrast no enhancement was noted. Features highly suggestive of PML

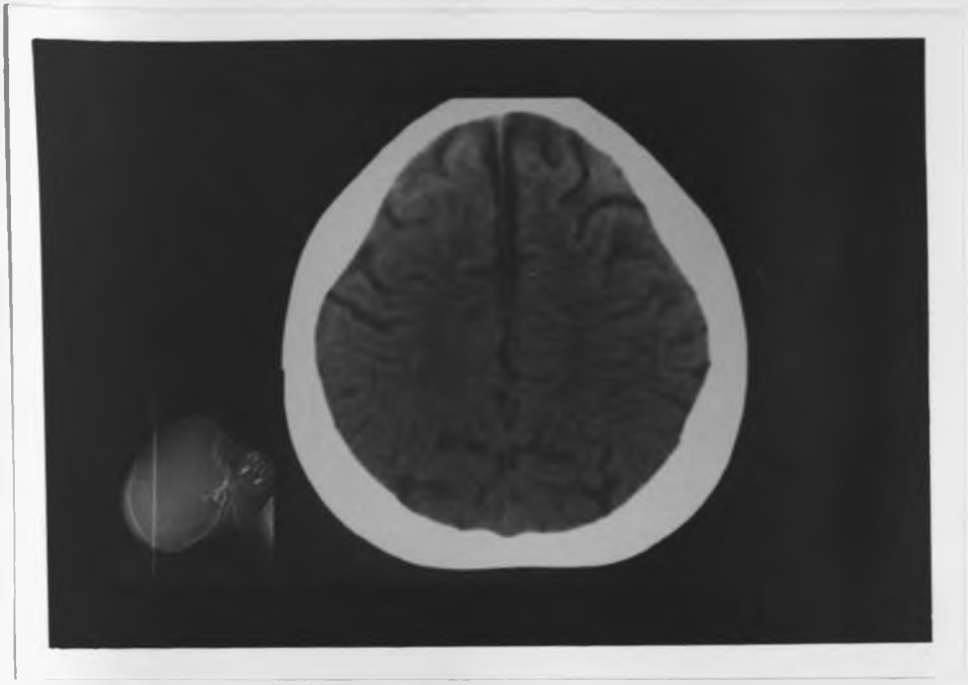


Fig 10 A

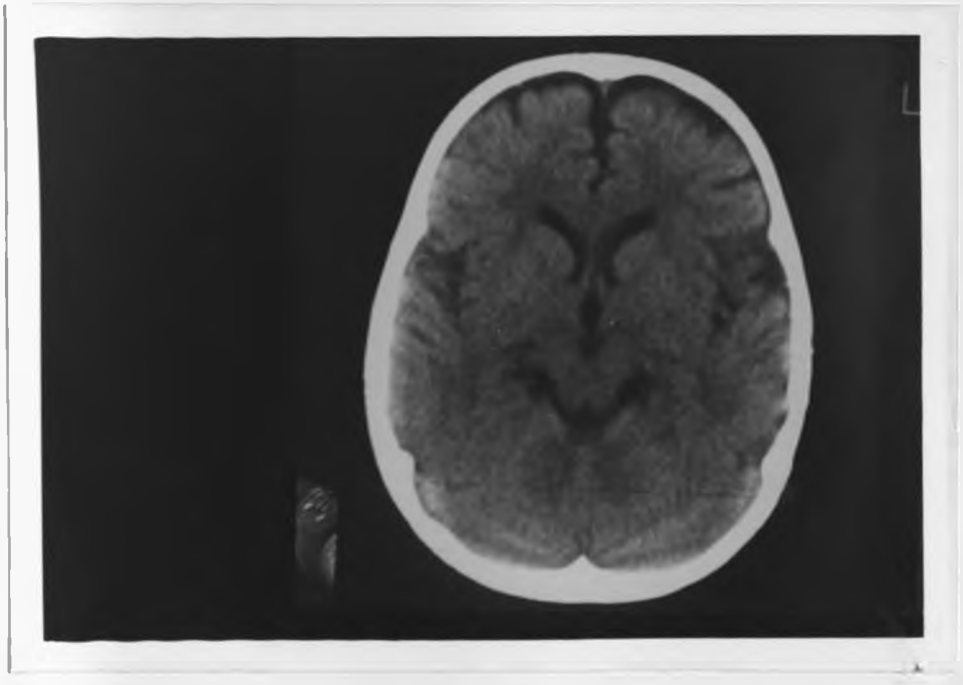


Fig 10 B

Fig 10(A&B)- Shows prominence of sulci; cisterns, interhemispheric fissure and ventricular system findings consistent with atrophy.

DISCUSSION

Sex Distribution

Out of 107 patients in the study 61 were male accounting for 57% and 45 were female accounting for 42.1%. The Male:Female ratio was 1:0.74.

There was a slight male preponderance though the study population was not large enough to give a conclusive sex ratio. Studies done elsewhere vary depending on the mode of initial introduction of virus.

Epidemiologic studies indicate that transmission of HIV-1 in Africa and Haiti is primarily by heterosexual a 1:1 sex ratio of HIV infection was noted (59). In areas where virus was introduced at an early stage among drug users the sex ratio of male:female AIDS cases is decreasing regularly "picturing" the progression of the spread among female, in N. America rates decreased from 10.7 :1 in 1987 to 6.5:1 in 1991, in Brazil it decreased from 9.7.1 in 1987 to 5.5:1 in 1991, in Europe it decreased from 7.5:1 in 1987 to 5.2:1 in 1991 (60). While in Australia the ratio was indicated to have remained high at 24:1 in 1987 to 31:1 in 1991 (61).

On other areas where introduction was by heterosexual contact, the sex ratio was close to 1:1 and no notable changes have been observed over time. Rates in Caribbean Islands reached 2.5. :1 in 1987 and 2:1 in 1991 (60).

In Africa where the virus was introduced in the heterosexual population and is still spreading in this population overall trends are not available. However, some sex ratio are available for some countries and it is thought to be slightly less than one. 0.8:1 in Kinshasa, 0.9:1 in Uganda (1989) whereas in Ivory Coast and Senegal men out number female patients by 2:1 and 4:1 respectively (69).

Age Distribution

The age scanned (see table II) ranged from 8 months to 52 years with a mean age of 33.42 years. The commonest age group of patient scanned was found to be between 31-40 years with a frequency of (36.4%) of the study population. This was followed by the group with age between (21-30) years, with a frequency of 23 (21.5%). The 3rd commonest group was between 41-50 years with a frequency of 22 (20.5%). Age group between (0-10) years had 7 patient accounting for (6.5%). Age group between (11-20) had 3 patients, while age > 50 years had only one patient.

Adult patients with ages between 21-50 years account for 88% of the study population. Epidemiological studies done elsewhere indicates that (80-90%) of the patients infected are the most sexually active age group between (20-40%) (59).

The national USA prevalence of AIDS varies with location between rural low levels to high urban levels of upto 130/100,000 in San Francisco, and in age (25-44) AIDS is listed as the 2nd commonest cause of death after trauma for male and fifth in female (63). In both extremes of age there were fewer patients. In age less than 10 years the frequency was 7(6.5%), while age more than 50 years there was only one patient. In the younger extreme the frequency was more when compared to the older extreme. This is because women in there reproductive age group are equally infected by the HIV especially by heterosexual spread, therefore mother to infant spread is a major problem. In some areas 5-15% or more of pregnant women are HIV infected. Between 25% to 50% of the babies born to these infected mothers will be infected with HIV (63).

Distribution of clinical presentation

In the distribution of clinical presentation, (see tables III, V and VII) majority of the patients were in group 5, which contained patients presenting with focal neurological signs. This group had a frequency of 43 patients which accounts for (40.2%) of the patients scanned and only 6 patients (5.6%) scanned in this group had normal CT finding. The group had the highest number of detectable radiological focal lesions when compared to the other groups. For example it contained 13 (40.6%) of the 32 patients with abscess, 4 (50%) of the 8 patients with tumour, 6 (40%) patients with infarcts, 6 (60%) out of 10 patients with features of encephalitis and 11 (36.7%) of the 30 patient with atrophy. From this we can deduce that

there was good correlation between patients with focal neurological complaints with detectable focal radiological findings, since 22 (52.3%) out of 43 patients in the group had radiological focal lesions. Though not all patients with focal clinical neurological signs had focal radiological findings and vice versa. This may be because AIDS patients may present with compounded clinical presentation such that some demonstrable radiological findings have been found in brains of patients with no clinical neurological complaints, as highlighted in the literature review.

Group 2 had the second commonest occurence where 19 patients accounting for (17.8%) were in the group. Patients in this group had various radiological findings. 7(23.3%) patients had brain atrophy, of these patients with atrophy 3 had atrophy alone and the rest had atrophy with other accompaning radiological findings. For example, I patient had atrophy and encephalitis, 2 patients had atrophy and infarct, 1 had atrophy, abscess and meningitis and one patient had atrophy infarct and meningitis. The commonest radiological findings in this group was brain atrophy.

Group 3 had the 3rd commonest occurrence. Where 15 patients (14%) presented with meningitis like symptoms. In this group majority of the patients had brain atrophy which was reported in 7 patients (23.3%). Other radiological findings included normal findings in 3 patients, meningitis in 3 patients (60%). Encephalitis, other, hydrocephalus and ?tumour each had one patient. For various radiological combination (see table VII).

In group 4 which consisted of patients presenting with fits, 14 patients (13.1%) were registered in the group. Most of the patients in this group had radiological features of abscess which was reported in 6 patients(18.8%), other radiological findings reported in the group included normal findings in 4 patients (21.1%), atrophy in 3 patients (6.7%), encephalitis in 1 patient and tumour 1 patient. There was a correlation between clinical neurological symptoms and radiological findings since half of the patients in this group had focal radiological findings explaining the occurrence of this.

Group 6 had 11 patients (10.3%), these patients presented with depressed level of consciousness. Majority of patients in this group had features of abscess which was reported in 5 patients (15%), 2 patient (6%) had atrophy and 2 (10.5%) had normal findings and one patient was reported to have encephalitis.

Group one had the least number of patients with 5 patients (4.7%). The patients in this group presented with persistent headache. 3 patients (9.4%) had abscess, one had atrophy and one patient had normal finding.

DISCUSSION OF CT SCAN RADIOLOGICAL FINDINGS

In the 107 patients scanned 19 (17.8%) had normal CT scan reports. While the rest 88 (82.2%) had some radiologically detectable abnormalities.

The accuracy of CT in detecting intracranial abnormality in AIDS was 82.2%. This emphasis the importance of CT in management of HIV patients with neurological symptoms.

The normal reports were given in 17.8%. This may be due to the insensitivity of CT in detecting early cortical lesions and especially when situated in the white matter, as highlighted in the literature review. Encephalopathy secondary to other organ failure may also depict a normal report.

Brain Abscess

Brain abscess was the commonest radiological abnormality reported. It was reported in 32 patients which accounts for (29.9%). Most of the abscesses were in multiple sites, since 20 patients (62.5%) who had reports of abscess had multiple lesions. Most of these lesions were found in the cortical regions of the brain (see table IX) either in the temporal, frontal, parietal and occipital lobe. There was slight preponderance to the parietal lobe. Other regions of the brain involved in multiple abscesses included the basal ganglia and thalamus.

Single abscess lesions were reported in 12 patients (37.5%). 7 cases were located in the cortical region while 3 lesions were in the basal ganglia and one in the thalamus.

On imaging abscess appeared as a hypodense lesion with surrounding brain oedema with or without shift of the surrounding structures and rim enhancement post contrast was noted. In 3 of the patients with abscess there was associated hydrocephalus secondary to obstruction of the CSF flow. Other associated imaging findings accompanied by abscess included atrophy in 3 patients. Encephalitis in 1 patient and in 1 patient meningitis (see table VI).

13 patients (40%) with abscess were in group 5 (see table V). The rest of the patients were distributed to the other groups; group 4 had 6 patients (18.8%), group 6 had 5 patients (15.6%), group 25 patients (15.6%), and group 1 with 3 patients (9.4%).

As emphasized in the literature review it is hard to differentiate radiologically between CNS lymphoma from an abscess. Therefore clinical history, physical examination

finding , laboratory finding and a chest radiograph if available may help the radiologist to make a better diagnosis. Biopsy and histology is the main stay of diagnosing lymphoma.

Atrophy

Atrophy was the second commonest reported radiological finding. It was reported in 30 patients (28%) second to abscess 32 patients (29.9%) of the patients scanned.

On imaging atrophy is indicated by cerebral cortical loss, which is marked by prominence of the sulci, interhemispheric fissure and ventriculomegally. From the study (see table V), most patients with atrophy were in group 5 of clinical presentation, which registered 11 patients (36.7%). The other patients were distributed in the other group where group 2 and 3 each had 7 patient (23.3%) group 4 and 6 each had 2 patient (6.7%) and group 1 had 1 patient.

Patients with atrophy alone were 17 patients, (see table VII). The rest of the patients had a contribution of atrophy with other radiological findings e.g. atrophy and abscess 2 patients, atrophy and encephalitis 3 patients, atrophy and infarct 5 patients. Atrophy, abscess and meningitis one patient and atrophy, infarct and meningitis one patient. Studies done elsewhere show that atrophy is the most frequent radiological finding in HIV patients (64).

This study shows that abscess was the most common radiological finding. On imaging abscess could mask the features of associated atrophy because of the oedema and Midline shift which commonly accompany the pathology.

Infarcts

Radiological features suggestive of infarcts occurred in 15 patients (14%). Majority of these patients were in group 5 of clinical presentation (see table VI) with 6 patients accounting for 40% of patients with infarcts. 5 (33.3%) patients were in group 2, 3 patients (20%) in group 3 and one patient (6.7%) in group 6. 8 lesions were in the cortex, while 7 infarct lesions were in the basal ganglia, (See table IX).

On imaging infarcts were depicted as low density lesions precontrast and post contrast some lesions did not enhance, others showed evidence of re-vascularization. On the study as shown in (table VI), 6 patients had infarct with no other associated radiological findings, while the rest of the patients had other accompaning radiological abnormalities e.g. infarct and atrophy occurred in 5 patients, infarct and encephalitis in one patient, infarct and

other (Sinusitis) in one patient, infarct, atrophy and meningitis in one patient and infarct meningitis and hydrocephalus in one patient.

The infarcts may be related to infections e.g. as seen in meningitis or may be secondary to other causes e.g. atheroma or vasculitis as indicated in the literature review.

One patient was reported to have a bleed in the right thalamus. From the clinical history the patient was reported not to be responding to anti-meningitic treatment.

Encephalitis

Encephalitis was reported in 10 patients (9.3%), (see table IV). Majority of these patients were in group 5. Radiologically they were small multifocal hypodence lesions in the cortex and cortical medullary junction with no associated mass effect and post contrast they didn't enhance. In 3 patients with encephalitis brain atrophy was also reported (see table V). One patient was reported to have encephalitis and abscess, encephalitis and infarct was also reported in one patient. The small multifocal hypodense lesions are due to demyelinative effect induced by HIV or may be secondary to other viruses e.g. papova virus and CMV. The papova virus leads to progressive multifocal leucoencephalopathy (PML) which was reported in one patient in the study. In PML the small multifocal demyelinative lesions progress over several months to larger bilateral confluent areas.

In the earlier stage of acute or sub acute encephalitis the focal lesions might not be detectable by the imaging modalities. The demonstration of microglial nodules with multinucleated giant cells is the hallmark of HIV encephalitis (65). Post (3), found that CT and MR usually misses or grossly underestimated the primary parenchymal abnormalities of HIV infections and postulated that the insensitivity is due in part to the microscopic size of the lesions and their diffuse, non focal, non mass producing nature and in part to the fact that they elicit little oedema, as opposed to many other AIDS related lesions e.g. Toxoplasmosis or primary CNS lymphoma.

?Tumours Mass

8 patients showed radiological features suggestive of ?CNS tumour, most likely lymphoma. The masses were hypodense precontrast. After contrast material administration irregular or rim like pattern of enhancement was noted. In 4 patients the lesions were situated in the basal ganglia, in 3 patients the lesions were in the parietal lobe and in one patient a CT myelogram was done and a destructive mass was noted to be arising from the

left iliopsoas and eroding and destroying the surrounding bones the sacrum and left iliac wing.

The differential diagnosis of these cranial tumours were lymphoma or abscess. As discussed in the literature review radiologically it can be very difficult to differentiate abscesses from CNS primary lymphomas. When available single photon emission tomography (spect) using thallium 201 may be used. Neoplasm like lymphoma will take the thallium and it will be shown as a hot sport, while abscess will be depicted as a cold area. Another imaging modality which can be employed to differentiate an abscess from lymphoma is MR spectroscopy. It has also been shown that Toxoplasmosis and lymphoma have highly distinctive chemical features. In Toxoplasmosis there is absence of normal brain metabolites, whereas an increase in choline may be observed in lymphoma. In our set up spect and MR spectroscopy are not available. So if in doubt the patient can be started on treatment for anti Toxoplasma for 2 weeks and follow up CT scan or MRI done to assess the radiological improvement. Clinical or radiological improvement then points to the diagnosis but when there is none then a brain biopsy is indicated.

Meningitis

5 patients (4.7%) were found to have radiological features suggestive of meningitis. Radiologically depicted by leptomeningeal enhancement post contrast. Of the 5 patients 3 had meningitis like symptoms of headache, neck stiffness and photophobia. The other 2 patients were in clinical group 2. Only 2 patients had basal enhancement alone. The other 3 had combination of radiological findings where one patient had brain atrophy and abscess, another patient had atrophy and infarct and the last patient had infarct and hydrocephalus. Meningitis usually presents with meningeal enhancement in the basal cisterns with or without hydrocephalus. In his study, Villoria (67) of the 35 patient with AIDS and proved intracranial tuberculosis found that hydrocephalus was present in 51% followed by meningeal enhancement. Hydrocephalus occurs secondary to obstruction of CSF flow.

Meningitis can be secondary to any of the opportunistic infections described in the literature review.

RECOMMENDATIONS & CONCLUSIONS

1. With the increasing incidence of AIDS and a large number of patients developing CNS complications. CT scanning has been found to be a good imaging modarity in detecting intracranial lesions in AIDS patients, and especially since most of the patients scanned in the study had radiological diagnosis of abscess early diagnosis and treatment will positively influence the outcome.

2. There is marked under utilization of CT scan in the follow up of AIDS patients with focal lesions in our set up since none of the patients scanned had follow up CT scan examination. Elsewhere CT scan follow up has been shown to be important in the management e.g. if the initial radiological diagnosis was reported as abscess and the lesion is not resolving or it is increasing in size then brain biopsy is usually indicated, since CNS lymphoma might present radiologically like an abscess.

3. For normal CT scan finding, if the patient has persistent neurological complains MRI may be recommended.

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APPENDIX

Data collection form.

1. Patients unit No/
2. Age
3. Sex
4. Complaints and Clinical findings
5. Use of contrast media
6. Radiological findings.