A DISSERTATION

ON

A STUDY ON CLINICAL PROFILE OF PAEDIATRIC HIV INFECTION IN THE AGE GROUP OF 18 MONTHS TO 12 YEARS AND TO CORRELATE WITH CD4 COUNT

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CERTIFICATE

Certified that this dissertation entitled **"Study on Clinical Profile of Paediatric HIV Infection in the age group of 18 months to 12 years and to correlate with CD4 count"** is a bonafide work done by **Dr. R. SURESHKUMAR M.D** postgraduate in **PAEDIATRIC MEDICINE** at Government Mohan Kumaramangalam Medical College, Salem, during the academic year 2007 to 2009.

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DECLARATION

I declare that this dissertation entitled **"Study on Clinical Profile of Paediatric HIV Infection in the age group of 18 months to 12 years and to correlate with CD4 count"** done by me at Government Mohan Kumaramangalam Medical College Hospital, Salem under the guidance and supervision of my department chiefs **Prof. K.MUTHUKUMAR, Prof. R. SIVAGAMASUNDARI, Prof. M.RATHINASAMY.** It is submitted in part of fulfillment of the award of the degree of MD (Paediatrics) for the March 2009 examination to be held under the TamilNadu Dr. MGR Medical University, Chennai. This has not been submitted previously by me for the award of any degree or diploma from any other university.

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CONTENTS

AIM

1. To study the clinical profile of pediatric HIV infection in the age group of 18 month to 12 years

2. To correlate the clinical features with the CD_4 count.

STUDY JUSTIFICATION

HIV / AIDS is a global epidemic now and it has the potential to affect all countries and all population[2].

Approximately 50% of new infection occur in the young adults <25 years[2] and if they left untreated, will die within 10 years of contracting the infection. Hence it becomes very important to identity these patients and put them under regular followup and to start ART, if necessary at the earliest.

India accounts for high global HIV burden and Tamilnadu comes under Intermediate prevalent zone with antental mothers with HIV infection <1% with STI > 20%[7].

As per the sentinel surveillance report by NACO for the year 2006-2007, incidence of HIV infection in antenatal mothers in Salem is 4% and hence mother to childhood transmission increases multifold[7].

This study is aimed to identity the early clinical features of HIV infection. Thus

by knowing the clinical spectrum of HIV infection in our region, screening can be intensified on the suspected children and early diagnosis can be made, which will help in early management and in decreasing the incidence of HIV related morbidity

MATERIALS AND METHODS

Nature of Study	:	Descriptive study
Study population	:	100 children who were registered in ART centre over a period of 1 year were taken
Period and Place of study	:	This study was conducted over a period of 1 year from Oct 2007-Oct 2008 at Govt. Mohan Kumaramangalam Medical College Hospital, Salem.
Inclusion Criteria	:	Children who got registered in ART centre in the age group of 18 months to 12 years, whose diagnosis in confirmed by Rapid antigen tests or ELISA were included
Exclusion Criteria	:	Children <18 months were not taken as the facility for making diagnosis by PCR was not available in our centre.

A special proforma was designed to record the following information:

- Demographic data
- History at presentation

- Clinical findings
- Nutritional status
- Developmental History
- Stage of disease
- Parental and sibling status
- Mode of transmission

Informed consent was obtained from the parent / guardian for registering the above data in prescribed proforma.

Pulmonary Tuberculosis was confirmed by doing;

- Complete Blood Count
- CXR
- Sputum examination / Resting Gastric Juice analysis
- Non responsiveness to conventional antibiotics
- History of contact
- Response to ATT

TB lymphadenitis was diagnosed based on Blood complete FNAC report.

- HIV encephalopathy was diagnosed based on clinical features, CSF analysis, Neruo imaging.
- Diagnosis made on the skin lesions which is found to be one of the common manifestation were confirmed by Department of dermatology.
- IAP classification (for weight) and Mclaren's classification (for height) were used to

classify Nutritional status.

INTRODUCTION

EPIDEMIOLOGY

World over 38.6 million people are living with HIV, follow of which 2.3 million i.e. 5.9% are children <15 years of age[1].

- In 2007 – 4,20,000 children were newly infected.
- Estimated children living with HIV / AIDS in India is 0.17 – 0.24 million (3.5-5% of people with HIV).
- Prevalence of HIV in pregnant mother is 0.6% - 0.7% (National).
- Adult prevalence is 0.36% (0.43% males and 0.29% females)
- Children account for 18% of total deaths due in AIDS i.e. 1 in every six AIDS death every year is a child, but they are < 1 in every twenty five persons getting treatment.

India is divided into 3 (three) zones of prevalence.

(a) High Prevalence States :

(HIV Prevalence in AN mother > 1%)

Maharastra, Karnataka, Andrapradesh, Manipur, Nagaland. Average Prevalence is 1.6%

(b) Intermediate prevalence (on medium prevalence states):

(AN prevalence < 1% High risk population in STI clinics >20%)

Tamilnadu, Gujarat, West Bengal. Average prevalence is 0.5% in AN mothers.

(c) Low prevalence States

(AN HIV prevalence < 1% High risk population is STI clinics <20%)

Rest of the states. Average prevalence is 0.2% in AN mothers.

In developing countries 95% of cases in children are due to vertical transmission from infected parents. Approximately 15-30% in non breast feeding population. 30-45% in Breast feeding population.

(Breast feeding increases the risk by 5-20%)

In the absence of effective coverage of PMTCT measures, 30% of the exposed babies get infected at birth.

The risk of transmission can be reduced to < 2% by

a) ARV prophylaxis to women during pregnancy / labour and to the infant in first week.
b) Elective Caesarean Section – prior to onset of labour and rupture of membranes)
c) Complete avoidance of Breast feeding.

TRANSMISSION AND PATHOGENESIS OF PAEDIATRIC HIV DISEASE

HIV Virus

- RNA Virus
- Belongs to the class of Retroviruses in the subgroup of Lentiruses.

- Two types HIV-1 and HIV-2
  - Both can cause clinically indistinguishable AIDS
  - HIV-1 is predominant virus world wide.
  - HIV-2 is less easily transmitted, period of initial infection and illness is longer. This type is concentrated in West Africa.

- HIV-1 can be divided into three groups
  a) M (Major Group) – 90% of HIV belong to this category
  b) O (Outlier Group) – Restricted the West Central Africa.
  c) N (New Group) – Discovered in 1998 in Cameroon extremely rare.

Group M – has 9 clinically distinct subtypes:

A, B, C, D, F, G, H, J, K. HIV-1 Group M (C) is prevalent in India.

HIV STRUCTURE:

HIV virus is surrounded by a coat of Fatty material and forms **Viral envelope**.

gp120 / gp41 are projecting from the surface. Just below the envelope is a layer called **Matrix** made from protein P17. **Viral core or capsid** is bullet – shaped and is made of protein P24.

Inside the core are three enzymes. Reverse Transnsciptase, Integrase, Protease and two identical strands of HIV RNA.

Transmission of HIV:

- HIV present virtually in all body fluids of affected individual.
- High concentration of free HIV virus / HIV infected cells present in seminal, vaginal, cervical secretions (Genital fluids) and blood.
- Saliva has low HIV (< 1 particle/ml) with non specific inhibitory substances like fibroblasts and glycoprotein.
- Urine, Sweat, Amniotic fluid, Synovial fluid, faeces, tears have zero to few particles.

Route of transmission

- Vertical transmission
- Blood and Blood products
- Unprotected sexual contact.

Rate of Transmission

A) Vertical (> 90%)

Without intervention, overall transmission rate is

16-20% in developed countries.

25-45% in developing countries.

Due to

a) Breast feeding

b) High incidence of prematurity

c) Under nutrition among mothers

Estimated risk of MTCT

Age	Risk of transmission
0-14 weeks	1%
14-36 weeks	4%
30 weeks till labour	12%
During labour	8%
0-6 months (Post Natal)	7%
6-24 months	3%
Overall risk of transmission without breast feeding	20-25%
Overall risk of transmission with breast feeding till 6 months	25-30%
Overall risk of transmission with breast feeding upto 24 months	30-35%

Timing and Mechanism of transmission

Transmission occurs through transplacental transfer of virus (micro-circulation) during pregnancy, at the time of labour and delivery – due to exposure of skin and

mucus membrane to infected blood and cervico-vaginal secretion.

Transmission through breast feeding 70% occurring during first 4-6 months of feeding and longer the duration, higher the risk. Mixed feeding increases the risk.

Risk factors for Vertical Transmission:

Pregnancy	Labour & Delivery	Breast Feeding
High viral load	High viral load	High viral load
Placental infection	Rupture of members for more than 4 hours	Duration of breast feeding
STI	Invasive techniques that increase contact with mother's infected blood (Episiotomy, Fetal scalp monitoring)	Breast abscess, fissures, mastitis
Maternal Malnutrition	First infant in multiple birth	Poor maternal nutritional status
Maternal bleeding	Acute chorioaminionits	Oral disease in infant (thrush or Sores)
	Vaginal delivery	
	Prematurity	

B) Blood Borne Infections:

3-6% of Paediatric infections.

C) Sexual Transmission:

- Uncommon is paediatric population
- Happen from sexual abuse

- Receptive partners are at a high risk than ‘insertive’ patients
- Viral load determines the risks.

Risk of Transmission of HIV with different Routes

Sl. No.	Route of Exposure	Transmission rate	% of HIV infected individual who have acquired infection through this route	
			World	India
1.	Blood transfusion	90-95%	5%	7.05%
2.	Perinatal	20-40%	10%	11%
3.	Sexual Lute course	0.1-1%	75%	74.15%
	Vaginal		60%	
	Anal		15%	
4.	IV drug use	0.5-1%	10%	7.3%
5.	Needle Stick exposure	<0.5%	0.1%	0%
6.	Others			10.92%

PATHOGENESIS

HIV

- Infection leads to progressive depletion of CD4 cells and immune suppression.
- Infects cells other than T-cells, which express CD4
    - Monocytes and tissue Macro phages
    - Microglial cells of brain
    - Hofbauer cells of placenta.
    - Dendritic (Follicular dendritic cells in LN Langerhan cells in skin)
- Lymphonodes are the major reservoirs in asymptomatic HIV individuals.

HIV Replication

Can replicate only inside Human cells.

HIV attaches to CD4 through gp 120 and gp 41

Contents of the HIV particulars are released to the cell leaving the envelope.

HIV RT converts RNA to DNA.

This viral DNA spliced into Human DNA after curing the nutrition by HIV enzyme Integrase.

(This integrated HIV DNA – Provirus)

Provirus forms Messenger RNA

↓

New HIV proteins are produced

↓

Enzyme protease chops long strands of protein into smaller pieces forms new viral particles

↓

Released from the cell

Primary HIV ~~Infection~~

Via Mucosal exposure

↓

Mucosal dendritic cells bind HIV Envelope gp 120

↓

After processing, transport and present HIV to cells expressing

Via infected blood and blood products

↓

Directly go to lymph nodes And lymphoid tissue.

CD4 molecules.

↓

Then transport to regional LN and other

Lymphoid tissues, where intense replication

Occurs

When replication reaches critical level, burst of Viremia followed by rapid discrimination occur (usually 3-6 weeks after infection)

↓

Flu like illness – **Acute HIV Infection**

(observed is 50% of infected)

Clinically : Fever, rash, arthralgia, Lymphadenopathy

Lab : Significant fall in CD4, High Viral load 10^6-10^7 copies/ml.

↓

CD8 plays important role in containing infection Neutralizing antibodies appearing later help in continuing the suppression of infection.

↓

CLINICAL LATENCY

Latent Period / Clinic and Persistent Infection

- Acute viral disease disappear.
- Free and cell associated virus decreases.
- CD4 near normal.
- Neutralizing virus specific antibodies increases.
- Chronic and persistent infection present inspite of intense cellular and humoral immune response.
- These high antibodies are not protective and do not interfere with cell-cell transmission
- This period lasts for 8-10 years in adults and much less for children.

Advanced HIV Disease

- Reemergence of the virus is peripheral blood and lymphnodes. Progressive decline in CD4 cell population. Loss of CD8 cell anti-HIV activity.
- Results in
    - Profound immune suppression.
    - End organ dysfunction
    - HIV associated opportunistic infections
    - Malignancies
    - Change in viral phenotype from Non syncytium inducing (NSI) to

Syncytium inducing (SI) virus.

Natural History of Pathogenesis of HIV-2

(i) Fewer immunological disturbance

(ii) Low transmissibility

(iii) Longer incubation period

(iv) Lower rate of vertical transmission

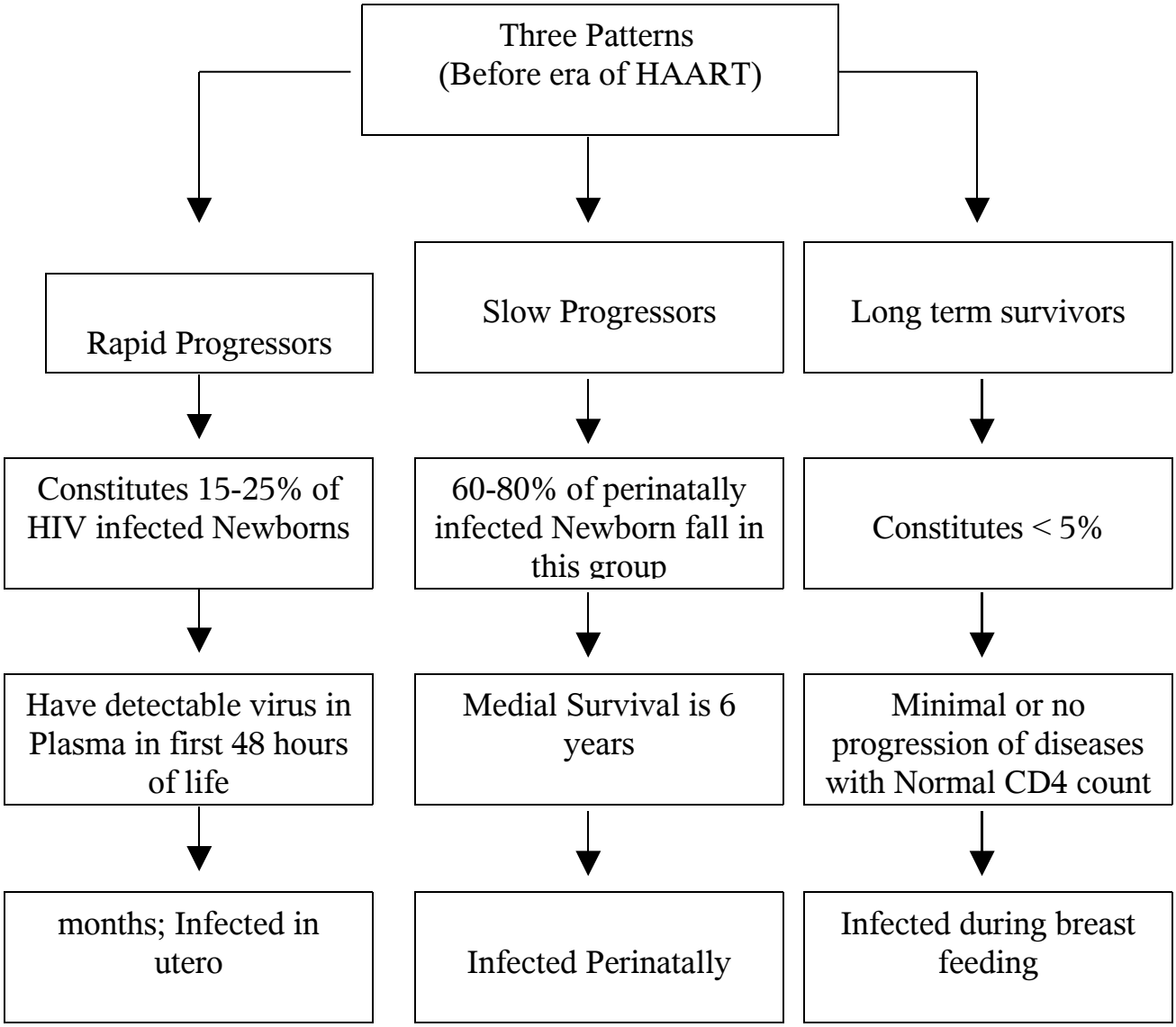
(v) Lower plasma TNF α

(vi) Lower Viral load

(vii) Lower level of Proviral DNA in circulating lymphocytes.

HIV-2 has a natural resistance to NNRTI – ARD, therefore not recommended

Natural History and Pathogenesis of HIV-1 (in Children)



CLASSIFICATION AND CLINICAL FEATURES

Two Classification

i) CDC Classification – Symptomatic HIV divided into four categories based on the severity of the symptom.

ii) WHO – Clinical classification immunological classification based on CD4.

Revised WHO Clinical Staging of HIV/AIDS for Infants and Children with Confirmed HIV Infection (> 18 months of age – HIV Antibody Test Positive, < 18 months of age – Virologic Test Positive)

Clinical Stage 1 (Aysmptomatic)

- Asymptomatic
- Persistent generalized lymphadenopathy

Clinical Stage 2 (Mild)

- Unexplained persistent hepatosplenomegaly
- Papular pruritic eruptions
- Extensive wart virus infection
- Extensive molluscum contagiosum
- Fungal nail infections
- Recurrent oral ulcerations

- Unexplained persistent parotid enlargement
- Lineal gingival erythema
- Herpes zoster
- Recurrent or chronic upper respiratory tract infections (otitis media, otorrhoea, sinusitis, tonsillitis)

Clinical Stage 3 (Advanced)

- Unexplained moderated malnutrition not adequately responding to standard therapy
- Unexplained persistent diarrhea (14 days or more)
- Unexplained persistent fever (above 37.5° C intermittent or constant, for longer than one month)
- Persistent oral candidiasis (after first 6-8 weeks of life)
- Oral hairy leukoplakia
- Acute necrotizing ulcerative gingivitis/periodontitis
- Lymph node TB
- Pulmonary TB
- Severe recurrent bacterial pneumonia
- Symptomatic lymphoid interstitial pneumonitis
- Chronic HIV-associated lung disease including bronchiectasis
- Unexplained anemia (<8g/dl), neutropenia (<0.5x$10^9/L^3$) or chronic

thrombocytopenia (<50 x $10^9/L^3$)

Clinical Stage 4 (Severe)

- Unexplained severe wasting, stunting or severe malnutrition not responding to standard therapy
- Pneumocystis pneumonia
- Recurrent severe bacterial infections (e.g. empyema, pyomyositis, bone or joint infection, meningitis, but excluding pneumonia)
- Chronic herpes simplex infection; (orolabial or cutaneous of more than one month's duration or visceral at any site)
- Extrapumonary TB
- Kaposi sarcoma
- Oesophageal candidiasis (or cnadidiasis of trachea, bronchi or lungs)
- Central nervous system toxoplasmosis (after one month of life)
- HIV encephalopathy
- Cytomegalovirus infection: retinitis or CMV infection affecting another organ, with onset at age over 1 month.
- Extrapulmonary cryptococcosis (including meningitis)
- Disseminated endemic mycosis(extrapulmonary histoplasmosis, coccidiomycosis)
- Chronic cryptosporidiosis
- Chronic isosporiasis

- Disseminated non-tuberculous mycobacteria infection
- Cerebral or B cell non-Hodgkin lymphoma
- Progressive multifocal leukoencephalopathy
- Symptomatic HIV – associated nephropathy or HIV- associated cardiomyopathy.

DIFFERENCES IN PEDIATRIC AND ADULT HIV- INFECTION

- Overall progression of disease is more rapid in children
- Immune system is more immature with higher CD4+ counts
- Recurrent invasive bacterial infections are more common in children.
- Disseminated CMV, Candida, Herpes Simplex and Varicella Zoster are more common.
- LIP occur almost exclusively in children.
- CNS infections are common.
- Peripheral neuropathy, Myopathy are rare in children.
- Kaposi Sarcoma is hardly ever reported.

The manifestations of the infection vary widely among infants, young children and adolescents. Most infected children are asymptomatic and do not have any abnormal findings on clinical examination at birth. The initial manifestations of the disease are mild and non-specific, these include lymphadenopathy, chronic or recurrent diarrhea, failure to thrive, wasting and oral trush. In contrast, the presentation in adolescents is similar to that in adults.

Common Manifestations of HIV-infection in Children

Manifestations MORE Commonly Encountered	Manifestations LESS Commonly Encountered
Lymphoid Interstitial Pneumonitis (LIP) Chronic Parotid Swelling **Opportunistic Infections:** • Tuberclosis • Pneumocystis Carinii Pneumonia • Serious Recurrent Bacterial Infections **HIV Encephalopathy**	**Kaposi's Sarcoma** **Malignancies:** CNS lymphoma **Opportunistic Infections:** • Cryptococcosis, • Histoplamosis, • Toxoplasmosis, Disseminated Mycobacterium Avium Complex (MAC) infection.

CLINICAL MANIFESTATIONS

Failure to Thrive

Failure to thrive is a very common feature of Pediatric HIV-infection in India. HIV-infection has an adverse effect on the growth of infected children and parameters such as weight, height and head circumference show deviation from the normal. The retardation of growth can be seen as early as 4-6 months of age in perinatally infected children. The factors that seem to contribute to growth retardation in HIV-infected children are many (i.e. low birth weight, decreased energy intake, diarrhea,

malabsorption, chronic diseases of the heart, kidney and lungs, micronutrient deficiencies, neuroendocrine abnormalities and repeated episodes of infection). Infants born to HIV-infected mothers have early growth delay, which have been correlated with high viral load in mothers. Length/height and weight are two anthropometrical parameters that represent growth in children. Therefore it is necessary that these parameters be regularly monitored in children, so that the earliest sign of growth retardation can be detected.

Hepatomegaly

Hepatomegaly is a common manifestation of pediatric HIV disease. It is likely to be caused by the replication of the virus in the reticuloendothelial tissue of the liver. Development of hepatomegaly within 3 months of age (in perinatally acquired HlV-infection) has prognostic significance since it is known to be associated with rapid progression of the disease. Malnutrition, fatty liver, steatosis, drug administration, CMV infection and malignancy can be some of the other causes for hepatomegaly in the HIV-infected children. Abnormal liver functions are common. Co-infection of Hepatitis C with HIV is well known.

Lymphadenopathy

HIV, after initial viraemia, gets trapped in the lymph nodes. The virus resides in the lymph nodes and also multiplies there. The replication of HIV in the nodes is the primary cause of lymphadenopathy. Other important conditions commonly associated

with HIV-infection that can give rise to generalized lymphadenopathy in HIV-infected children include tuberculosis, disseminated infections with other mycobacterial species, viral infections with CMV, Epstein-Barr virus (EBV) and malignancies like Iymphoma and Lymphosarcoma.

Chronic Diarrhoea

Diarrhoea is a very common clinical manifestation of HIV-infected infants and children in developing countries. As with many other symptoms of HIV- infection, the underlying mechanisms for diarrhea are many. These include infections, other inflammatory processes and malabsorption of carbohydrate and fats. HIV is presumed to be responsible for diarrhoea if one fails to isolate any organism. It is believed that HIV replication in the intestinal mucosa can lead to diarrhoea itself leading to HIV entropathy. Other causes of chronic diarrhea are opportunistic enteric infections with common organisms like Candida, Cryptosporidium, Cytomegalovirus, Giardia, Isosporabelli and Salmonella. These result in a dysfunctional immunity at the lamina propria of the gut leading to decreased secretary IgA, the principal protective immunoglobulin. The reduced helper T-cel! function results in the decrease of mucosal immune system of the gut as result of persistent infection, leading to prolonged diarrhea with malabsorption and malnutrition.

Infectious Agents Implicated as The Causes Of Diarrhoea In HIV-Infected

Children

Agent	Organisms
Viruses	Rotavirus, Cytomegalovirus, Adenovirus, HIV, HSV
Bacteria	Shigella, Camylobacter, E. coli, MAC, Salmonella
Protozoa	Cryptosporidium, parvum, Isosporabelli, Microsporidia, Entemoeba histolytica, Giardia Lamablia
Fungi	Histoplasma

Parotitis

Parotitis occurs as recurrent or chronic hypertrophic parotitis. The parotid swelling evolves gradually in-patients with HIV disease. It may be due to the direct infection of the parotid gland by HIV or due to lymphocytic infiltration of the gland. The swelling may be unilateral or bilateral but is classically painless and recurrent.

Skin Manifestations

HIV disease in children is associated with a number of skin manifestations. These can be due to infectious or non-infectious causes. Viral, bacterial and fungal infections have been very frequently reported in HIV-infected children. These usually tend to be more severe and resistant to therapy. Common skin diseases may present with unusual skin lesions such as Norwegian Scabies, disseminated, confluent and large lesions of Molluscum contagiosum.

SKIN MANIFESTATIONS IN HIV-INFECTED CHILDREN

Infectious Disorders and lesions	Non - Infectious Disorders and lesions
Viral Infections: Herpes simplex, Herpes zoster, Molluscum contagiosum, Warts	Seborrheic dermatitis, Atopic dermatitis CMV, Generalized dermatitis, Nutritional Deficiency
Fungal Infections: Candida, Tinea, Onchomycosis	Eczema, Psoriasis, Drug eruptions
Bacterial:	Impetigo Vasculties
Other:	Scabies Alopecia

Oralcandidiasis

Oral Candidiasis is the most common form of fungal infection encountered with HIV-infected children. It progresses to involve the esophagus in 20% of cases and denotes significantly impaired T-cell function, presenting with symptoms as anorexia, dysphasia, vomiting and fever. Other oral manifestations included in differential diagnosis are gingivostomatis, apthous ulcer, herpes labial peritonitis and oral hairy leuco plakia.

Hematological Abnormalities

Pediatric HIV disease is associated with different hematological abnormalities presenting as pallor, Neutropenia, Lymphopenia, Thrombocytopenia and Eosinophilia. Thrombocytopenia can be present with petechiae and ecchymosis and may be diagnosed as immune Thrombocytopenia. Alteration of hematological profile occurs due to the

virus itself, opportunistic infections, drugs side effects or antibody mediated cellular destruction.

Hematological Abnormality	Mechanisms Responsible
Anemia	Auto-immune antibodies that cause destruction of erythrocytes; Suppression of the bone marrow by drugs used in the treatment of HIV-infection (e.g. AZT) or of associated infections (i.e. ganciclovir, co-trimoxazole); Nutritional deficiency (folic acid, Vitamin B12, micro-nutrients)
Thrombocytopenia	Immune-mediated destruction of platelets, Nutritional deficiency (i.e. Vitamin B12 deficiency)
Neutropenia	Immune-mediated destruction of leukocytes
Lymphopenia	Bone marrow suppression due to altered cytokine production, CD4+ apoptosis induced by HIV replication
Eosinophilia	Shifting of immune response from Th1 toTh2 cytokine profile

Cardiac Manifestations

The perinatally infected children do not have a high risk of developing congenital cardiac malformations However, cardiovascular diseases do seem to develop in HIV-infected children, and they are often clinically silent. The cardiac manifestations of diseases noted in HIV-infected children include cardiomegaly, congestive cardiac

failure, non-bacterial thrombotic endocarditis, cardiomyopathy, pericardial effusion, cardiac tamponade, conduction disturbances and sudden death. Cardiomyopathy is frequently present in-patients with encephalopathy. The factors that have been implicated in the causes of cardiomyopathy include primary HIV disease, immune-mediated reactions, intercurrent infections and drug-toxicity. Cardiomyopathy decreases the survival rates and is one of the clinical indicators of starting ARV drugs.

Neurological Manifestations

Primary CNS infection by HIV is quite common as it is a neurotropic virus. Neuronal damage is mediated via astrocytes and neurotoxic cytokines. Neuronal dysfunctions through loss of supporting growth factors, excitotoxicity due to dysregulation of neurotransmitter re-uptake and increased permeability of the blood brain barrier, permit further seeding of the virus.

Two forms of Encephalopathy exist - Progressive and Static. HIV leads to a myriad of CNS problems of varied etiology, that are both infectious and non-infectious.

HIV Encephalopathy can cause developmental delay, with slow attainment of skills or regression of milestones. Developmental delay in HIV-infected children that begins in infancy can also be due to congenital opportunistic infections such as Toxoplasmosis, CMV or Rubella infection rather than due to HIV-infection alone. It

should also be appreciated that, HIV-infected infants are exposed to additional risk factors such as low birth weight, poor prenatal care, maternal illness, drug abuse and maternal death that can contribute to developmental delay. The developmental delay is more pronounced in the areas of gross motor, fine motor and language skills.

HIV Encephalopathy can also have manifestations such as loss of previously acquired milestones, cognitive impairment, neuropsychiatric problems, weakness, spasticity hyperreflexia, abnormal extra-pyramidal signs and cerebellar dysfunction. Problems such as learning disorders, memory and perception deficits have been described in asymptomatic or mildly symptomatic HIV-infected children as well. Distal peripheral neuropathy is a rare and recently described manifestation of HIV-Infection in children that is present with distal parasthesiae or pain, diminished ankle jerks and diminished vibration sense. The symptoms are usually mild and are chronic or fluctuating. Emotional liability, hyperactivity and lethargy are late signs of encephalopathy. HIV Encephalopathy usually manifests in the presence of significant immunosuppression and features of HIV disease such as Hepatosplenomegaly and Lymphadenopathy are usually present. It carries a poor prognosis. It may be emphasized that neurological manifestations like convulsions and alteration of sensorium can also be due to opportunistic infections that occur with greater frequency in these children. Tuberculous meningitis. Cryptococcal meningitis, pyogenic meningitis and CNS toxoplasmosis can give rise to neurological manifestations in H!V-infected children.

According to the CDC revised system, the diagnosis of Encephalopathy requires one of the following progressive findings to be present for at least 2 months in the absence of other identifiable causes:

- Failure to attain or loss of development milestones or loss of intellectual ability, verified by standard developmental scale or neuropsychological tests.
- Impaired brain growth or microcephaly demonstrated by head circumference measurements or brain atrophy demonstrated by CT or MRI, with serial imaging required in children less than 2 years of age.
- Acquired symmetric motor deficit manifested by two or more of the following paresis, pathological reflexes, ataxia or gait disturbances.
- CT may show evidence of cortical atrophy, prominent sulci, enlarged ventricles and decreased attenuation of the white matter suggestive of progressive multifocal leucoencephalopathy.

Nephropathy

The proportion of HIV-infected children who have nephropathy is variable and is more in adolescent. The manifestations of renal disease associated with AIDS include proteinuria, hematuria. hypertension, renal tubular acidosis, acute renal failure and progression to end-stage renal disease. In the initial stages, the patient could be asymptomatic, although laboratory evidence of nephropathy can be found on

investigations. Histological changes in AIDS nephropathy reveal focal segmental glomerulosclerrosis, minimal lesion glomerulonephritis, IgA nephropathy. Nephropathy may be due to direct infection of the virus, immune complex mediated vasculitis, or as a result of various opportunistic infections or drugs.

Respiratory Manifestations

Lymphoid Interstitial Pneumonitis (LIP) and Pulmonary Lymphoid Hyperplasis are the main presentations of the respiratory system involvement. The clinical presentation of these disorders are usually insidious. Cough and Tachypnea are early clinical features, followed by Dysprea at rest and development of clubbing. There are often no findings in the chest unless, recurrent bacterial infections have led to bronchiectasis. Hepatosplenomegaly, lymphadenopathy and parotid swelling are the commonly associated findings. The findings of diffuse bilateral reticulondular or interstitial infiltrates on chest radiograph ortomogram support the diagnosis of LIP. The nodules are usually 1-5mm in diameter and hilar lymphadenopathy is frequently present. A presumptive diagnosis is made on the basis of clinical and radiological findings. However, one needs to exclude the presence of opportunistic bacterial, viral and mycobacterial infections that can be present with similar clinical and radiological findings. Definitive diagnosis requires lung biopsy. Children with LIP have an increased incidence of pulmonary bacterial infections.

Tuberculosis is the most common opportunistic infection encountered in

children, occurring at a higher CD4 count when the immune-deficiency is comparatively less advanced- Mycobacterium avium inter cellular (MAC) causes disseminated and usually non-pulmonary disease and is an important cause of morbidity in HIV-infected children. A CD4 count <100 cells/mm3 has been recognized as a primary risk factor for this infection. Respiratory Viruses like Respiratory syncytial virus, Measles, Parainfluenza, Influenza, Adenovirus, Rhinovirus and Coronovirus, cause prolong and sever disease. Fungal infections due to Histoplasmosis. Coccidiodomcosis and aspergillosis present similarly. Pneumocystis Carinii Pneumonia (PCP) causes an acute life threatening pneumonitis.

Malignancy

The incidence of malignancy in HIV~infected children is higher than that in the general population. However, the type of malignancies associated with childhood AIDS (Non-Hodgkin's lymphoma, leiomyomas and leiomyosarcomas and leukemia) is much different from that associated with adult HIV disease. The Kaposi's sarcoma, a commonly encountered malignancy in adult HIV disease features is rare in children

Prognostic Indicators

In underdeveloped countries the age at diagnosis and the type of clinical presentation are the only clinical factors related to prognosis. Infants who develop symptoms in the first year of life manifest the fastest progression of illness with worst outcome. Similarly, the occurrence of opportunistic infections, progressive

encephalopathy or hypogammaglobulinemia at any age often carries a poor prognosis. In contrast, generalized lymphadenopathy, hepatosplenomegaly, parotitis are associated with a more favorable outcome.

Viral load is the most important prognostic marker of the risk of progression. But the availability and the cost are the constraints. It is predicted that a favorable clinical outcome is most likely if virus replication is maximally suppressed before the immune system is irreversibly damaged.

OPPORTUNISTIC INFECTIONS IN HIV / AIDS

Pneumocystis carinii (Jiroveci) Pneumonia

PCP is the most common OI associated with HIV in children and thus, it is also known as “AIDS pneumonia”. It is an infection of early infancy and predominantly occurs at the age of 3-6 months.

P. Carinii, a protozoan, is closely related to fungi. PCP occurring in immunodeficient HIV – infected infants is likely to represent primary infection. The reservoir and the mechanism of transmission of infection remain obscure. P.Carinii establishes as an extra-cellular parasite. Interstitial edema, hyaline membrances and proliferating organisms fill the air spaces, resulting in progressive hypoxemia and respiratory failure.

Children with PCP often present with a tetrad of Tachypnea. Dyspnoea, cough and fever. Physical examination reveals Tachycardia, respiratory distress, accelerating Tachpnea and diffuse retractions. Auscultation does not reveal any characteristic findings. ABG shows changes of hypoxemia, and respiratory failure.

The diagnosis can be confirmed by Wright-Giemsa staining of induced sputum or samples obtained by Broncho-Alveolar Lavage (BAL). Trophozoits and intracystic sporozoits can be demonstrated in the stained specimens.

Tuberculosis

One of the most common HIV-related OI is tuberculosis in the high prevalent areas. HIV increase the susceptibility to both the primary infections as well as to reactivation of tuberculous-infection due to depressed cell-mediated immunity.

Fever, cough, and weight loss, night sweats and malaise are common clinical findings. Fxtrapulmonary disease may involve other tissues and organs as the central nervous system, lymph nodes and mastoid.

Problems in diagnosis of tuberculosis occur as the presentation occurs with

unusual features and extrapulmonary disease. It may be associated with a negative tuberculin test. Careful medical history with emphasis on history of contact with tuberculous patient is important. The Mantoux Test is considered positive if the induration is 5 mm or more. The chest radiograph shows features of lobar or multi-lobar infiltrates or diffuses interstitial disease and hilar adenopathy. Isolation of the acidfast bacilli is the gold standard for diagnosis.

Candida Infections

Severe oral candidiasis maybe that first clinical indication of HIV-Infection. Oral thrush and diaper dermatitis are common. Thrush is extensive and is relatively difficult to treat. Older children present with decreased oral intake and dysphagia, Esophageal candidiasis present with substernal or abdominal pain dysphagia and weight loss.

The lesions seen in oral candidiasis are typical and can be confirmed by observing pseudohyphae on a KOH-stained specimen.

LAB DIAGNOSIS

Test for HIV Infection

Ant- HIV antibody test	Antigen detection	Virus isolation / Detection of viral nucleic acid
Screening: Microwell ELISA test Rapid tests.	P24 antigen assay	Viral culture
Supplemental Tests: Western blot Line immunoassay Recombinant immunobloting assay		PCR tests (DNA / RNA)

Indications for ART

WHO Pediatric Stage	Availability of CD4 cell measurements	Age-specific treatment recommendation	
		<12 months	≥ 12 months
4[a]	CD4	Treat All	
	No CD4[b]		
3[a]	CD4	Treat All	Treat All, CD4 guided* in children with TB, LIP, OHL, Thrombocytopenia
	No CD4		Treat All
2	CD4	CD4 guided	
	No CD4	Do not treat	
1	CD4	CD4 guided	
	No CD4	Do not treat	

a. Stabilize any opportunistic infection prior to initiation of ARV Therapy.

b. Baseline CD4 is useful to monitor ART.

When to start ART in Children, agewise, using CD4 guidance*

< 11 months	CD4 < 1500 cells/mm^3 (<25%)
12-35 months	CD4 < 750 cells/mm^3 (<20%)
36-59 months	CD4 < 350 cells/mm^3 (<15%)
> 5 years old	Follow adult guidelines i.e start ART if CD4<350 cells/mm^3 especially if symptomatic initiate before CD4 drops below 200 cells / mm^3

OBSERVATIONS

- A Total number of 100 children affected by HIV infection were studied over a period of one year from October – 2007 – October 2008.
- There were 65 males and 35 females in the ratio of 185:1.
- 22 Children were in the age group of <4 years and 78 children in the age group of 4 to 12 years.
- Mother to child transmission accounted for the highest presumed mode of transmission 97 (97%).

  Two children might be affected due to Blood transfusion given during Acute illness 2(2%).

  One child was infected by unidentified route.
- 98 children were delivered by normal vaginal delivery, one child was delivered by emergency LSCS and for one child mode of delivery not known (Since she was an adopted child)
- Among Hundred children delivered, only one child received (? Neviripine) during peripartum period.
- 49% of children received Breast milk alone for first 4 month, 50% of children received both Breast milk and cow's milk and only one child was completely replaced milk substitute.
- Among 100 children who were infected by HIV infection

22% were asymptomatic

78% were symptomatic.

- Asymptomatic children were identified during routine screening done, as their parents were found to be seropositive.
- Symptomatic children were divided into four groups based on the WHO clinical staging 2006 – Based on the nature of pathological status.
- Among symptomatic children,

Stage I - 10

Stage II - 39

Stage III - 24

Stage IV - 5

CD4% was on the higher side in stage I and II diseases and it is lower for stage III and lowest in stage IV.

CD4 RANGE

Sl. No.	Stages	CD4 Range	CD4 Ratio
1.	Stage I	349 - 2263	18% - 35%
2.	Stage II	304 -2083	7% - 29%
3.	Stage III	81- 1277	7% - 32%
4.	Stage IV	10 – 227	6% - 14.5%

The observed clinical features in the study subjects were :

Skin lesions (23%), Neck swelling (17%), Respiratory symptoms and Recurrent RTI (16%), Oral lesions (10%), Failure to the (71%), GI symptoms (5%), Chronic eardischarge (5%), Recurrent febrile illness (19%), and Neurological symptoms (4%).

Skin lesions alone present in (9%) and along with other symptoms in (14%).

The clinical sings were,

PEM (76%) Generalised lymphadenopathy (46%), Skin lesions (29%), Anaemia (16%), Oral lesions (10%), Anaemia (16%), Chronic parotid swelling (2%), CSOM (6%).

On systemic examination,

Respiratory system involved in 14%.

Gastrointestinal system 3%

Central Nervous system 4%.

Lymphadenopathy is observed in (46%)

Generalised significant lymphadenopathy alone founding 12% of patients. TB lymphadenitis in 12% and lymphadenopathy with other clinical features in 22%.

CD4% was on higher side average being >25% in patient presented with lymphadenitis and average being 12-14% in those who had lymphadenopathy along with other stage II, III and stage IV disease.

Skin lesions being the second common finding which were observed in 29% of patients.

Pruritic papular Eruption was the commonest clinical skin lesion noticed in 14 children and the CD4% for this feature was ranging between (24-35%).

Scabies in 2 children, (CD_4%, 12-20%) Molluscum in 1 (CD_4 11%), Extensive impetigo in 2 (CD_4 11%) Herpes in 3 (CD_4 between 7-31%). These above mentioned four features were associated with low CD_4 count compared to other features mentioned in the table.

Oral lesions were found in 10 children

Oral candidiasis was observed in 3 children whose CD_4 range was between (8-10%), leukoplakia in 2 children (CD_4 25%). Glossitis and Gingivostomatits in 5 children average CD_4 count being 29% .

Chronic Suppurative Otitis Media

This was observed in 5% of children with CD4 count ranges between (7-19%).

Chronic parotid enlargement noticed in two children with the CD4 count 25% and 12%.

CLINICAL FEATURES

Clinical Features	No. of Cases	CD4 Ratio
Asymptomatic	22	18% - 35%

Lymphadenopathy - 46 cases

Clinical Features	No. of Cases	CD4 Ratio
Lymphadenopathy alone	12	18% - 35%
Along with other stage II and stage III clinical features	22	7% - 29%
TB Lymphadenitis	12	11% - 32%

Skin Lesions – 29 cases

Skin Lesions	No. of Cases	CD_4%
Scabies	2	12% - 20%
Eczema	4	20% - 24%
Molluscum	1	11%
Herpes	3	7-31%

Pruritic Papular eruptions	14	24-35%
Extensive impetigo	2	11%
Seborrhic dermatitis	3	22% - 24%

Oral lesions

Oral lesions	**No. of Cases**	**Average CD^4**
Candidiasis	3	8-10%
Leukoplakia	2	25%
Glossitis	2	30%
Gingivostomatitis	3	28%

Among the opportunistic infection in these children

Pulmonary tuberculosis was found in 13 cases, Disseminated TB 2 cases, TB Spine 1 case, TB meningitis 2 cases and TB lymphadenitis 12 cases. Thus Tuberculosis being the commonest opportunistic infection.

Oral candidiasis was observed in 3 cases with CD_4 ranges between (8-10%) .

Pneumocystic carinii pneumonia (suspected) was observed in one child with CD_4 count (11%).

Opportunistic Infections

	No. of Cases	Average CD4
1. Pulmonary TB 12 cases		
Pneumonia	10	7% - 31%
Empyema	1	17%
Bronchiectasis	2	7%, 16%

2. TB Spine	1	10%
3. Disseminated TB	2	9%, 11%
4. TBM	2	6%, 14.5%
5. Extrapulmonary TB	12	11% - 32%

6. PC Pneumonia (Suspected)	1	11%

On systemic examination in these children,

i) Respiratory system was involved most commonly (14 children) and TB pneumonia (Bronchopneumonia – 6 cases, Consolidation – 4 cases) was most common (10 children). CD_4 count of,

- TB pneumonia with CD4 count between (6 – 31%)
- Empyema was observed in one child (CD_4 17%)

- Bronchiectasis in 2 children CD_4 % (7% - 16%)

ii) Next was CNS involvement with CNS involvement with

- TB Meningitis 2 cases (CD_4 % 6% - 14.5%)
- TB spine in 1 child (CD_4 % 10%)
- HIV encephalopathy is child 9 (CD_4 – 7)

iii) GI system was involved in 3 children

- Chronic diarrhecn in one child (CD_4 %– 8%)
- Hepatosplenomegaly in two children (CD_4 9% - 10%)

All the study group 100 children were classified as per IAP classification as below

Grade stage I^{o} - 27%

Grade stage II^{o} - 24%

Grade stage III^{o} - 16%

Grade stage IV^{o} - 6%

Wt >80% as expected in 23%.

For height Mclaren's classification was used and the various categories were observed as below.

<80% of expected Height (stunted) in 12%

80-93% expected Height (short stature) in 55 %

>93 % expected Height considered as normal in33%.

NUTRITIONAL STATUS

Wt: (against expected wt)

Weight (%)	Stage I	Stage II	Stage III	Stage IV
< 50%	0	0	5	1
50-60%	2	6	6	2
60-70%	9	9	5	1
70-80%	10	12	4	1
> 80%	9	9	5	-

Ht: (against expected Ht)

Height (%)	Stage I	Stage II	Stage III	Stage IV
< 80%	1	4	5	2
80-93%	19	18	15	3
> 93%	13	15	5	0

SUMMARY

100 children who were infected with HIV were taken as study group. Among them,

Asymptomatic were 22%

Symptomatic were 78%

PEM observed in 76% cases and severe PEM was noticed (Grade III and IV and) in 22%.

Among the symptomatic HIV infected children the clinical feature that were observed,

Generalised lymphadenopathy – 46%

Lymphadenopathy alone observed in 10% with average CD_4 count being 25%.

Lymphadenopathy associated with other features of stage II to IV HIV infection like pneumonia, skin lesions, oral lesions, severe PEM constitute about 26% and the average CD_4 count in this group being 12-18%.

Generalised lymphadenopathy was the most common clinical manifestation in this study.

- Skin lesions were observed in 29% of children. Pruritic papular eruption being the

common finding observed in 14 children with average CD_4 % is bet (24 – 35%).

Other lesions like scabies (CD_4 11%), Herpes (CD_4 7 – 31%) molluscum (CD4 11%). Extensive impetigo (CD4 11%) were also observed. These lesions were associates with low CD_4 count.

- Among oral lesion that were observed oral candidiasis was found in 3% and associated with low CD4 count (8-10%)
- Among opportunistic infections, tuberculosis was found to be the commonest infection which was 30% (This include all forms of TB)
- Severe anemia (<8g%) was found in 16% which were predominantly in the children belong to stage III a stage IV illness, who were also associated with low CD4 count.
- As for as Nutritional status in concerned, children in stage IV disease belong to Grade III & IV PEM and height being <85% of the expected, where us, majority of stage I disease children have Grade IPEM.
- MTC transmission accounts for 97% and being the majority one.
- Only one was delivered by emergency LSCS and the rest were delivered by normal vaginal delivery. For one child mode of delivery not known.
- Only one child was replaced with milk substitute and given neviripine ? perinatally. Rest of the children were given either Breast milk or mixed (both BF & Cow's milk) not received Neviripine.
- Since children infected with HIV having, Asymptomatic generalised lympadenopathy, skin lesions like eczema, pruritic papular eruptions, oral lesions

like glossitis and gingivostomatitis were associated with the high CD4 count, intensifying the screening for HIV infection by high suspicion will help in diagnosing HIV infected children at the earliest, and thus they can be subjected to early management and follow up periodically. Early management include – chemoprophylaxis, immunization, management of opportunistic infection, nutritional support and ARV Therapy.

- Proper ART to AN mother who were identified as HIV infected, ART to mother and baby during peripartum period, Elective LSCS, proper milk substitution will definitely found to be effective in reducing MTC transmission.

LIMITATIONS

- In this study mostly we have observed clinical features of slow progressors and since children < 18 months were not included clinical manifestation of rapid progressors could not be identified.
- Diagnosis of PCP in one child was made on the basis of clinical features and a CXR finding. This was not confirmed by BAL / Biopsy.
- Investigations to identify CMV, toxoplasmosis, hepatitis infection were not done.

LITERATURE REVIEW

When we go through the various literature on the various Indian and non-Indian studies on the clinical profile of paediatric HIV, this study has some similarities and few differences from them.

As is all previous studies,

- Perinatal transmission was the most common mode of transmission in this study.
- Dermatological manifestation and generalized lympadenopathy were the frequent manifestation among the HIV infield children.
- Tuberculosis was found to be the commonest opportunistic infection in our country and this study also reflected that.
- Severe PEM (III & IV) was found in significant percentage in severe form of HIV infection.
- Hepatosplenomegnly was less in number compared to various Indian & non-Indian studies.
- Comparison of clinical profile of our study done with other Indian and Non-Indian studies given in the following tables.

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CLINICAL PROFILE OF HIV IN PAEDIATRIC AGE GROUP FROM 18 MONTHS TO 12 YEARS AND TO CORRELATE WITH CD4 COUNT

Name :

Weight :

Age / Sex :

Height :

Address :

Complaints :

	Yes	**No**
Fever	☐	☐
Cough	☐	☐
Diarrhea	☐	☐
FTT	☐	☐
Oral ulcers	☐	☐
Skin rash	☐	☐
Breathlessness	☐	☐
Seizures	☐	☐
Swelling in neck/axilla	☐	☐
None	☐	

Complaints :

	Yes	No
Oral Candidiasis	☐	☐
Chronic diarrhea	☐	☐
Recurrent RTI	☐	☐
Herpes Zoster	☐	☐
Blood transfusion	☐	☐
Seizures	☐	☐
Jaundice	☐	☐
Unsafe injection/surgery	☐	☐
Recurrent Febrile Episodes	☐	☐
None	☐	

Birth History:

Normal ☐ Caesarean ☐ Vaccum ☐ Forceps ☐

Birth Weight

Neviripine ☐ ☐

Feeding

Breast Milk ☐ Replacement ☐ Mixed ☐

Development

Normal ☐ Abnormal ☐

Examination :

Nutrition Normal ☐ Abnormal ☐

	Yes	**No**
Anaemia	☐	☐
Cyanosis	☐	☐
Fever	☐	☐
Tachypnoea	☐	☐
Jaundice	☐	☐
Pedal edema	☐	☐
Lymph nodes	☐	☐

Matted ☐ Discrete ☐

Cervical ☐ Axillary ☐ Others ☐

Skin Lesions:

TBA Scabres Herpes zoster

Seborrhic dermatitis ☐ taeniasis ☐ Warts ☐

Oral Vacity

Normal ☐ Abnormal ☐ (specify)

Parotid Swelling	☐	☐
Ear dischife	☐	☐
Petrechi / Purpura	☐	☐

Systemic Examination:

Abdomen

Normal ☐ Abnormal ☐

Respiratory Space

Normal ☐ Abnormal ☐

Cardiovascular System

Normal ☐ Abnormal ☐

Central nervous system

Normal ☐ Abnormal ☐

Investigations

Diagnosis :

HIV Status : Symptomatic ☐ Asymptomatic ☐

WHO Clinical State : I ☐ II ☐ III ☐ IV ☐

Parental Status : Father ☐ M☐er ☐

Sibling

Occupation :

Socio Economic Status :

CD4 Count :

Treatment :