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STUDY OF THE RELATIONSHIP BETWEEN CD4 COUNT AND CLINICAL FEATURES IN HIV – INFECTED PATIENTS IN SOUTH INDIAN POPULATION

***Vinay KV¹, Sandeep GN², Vishal K³ and Beena DN⁴**

^{1,3} Department of Anatomy, K.S. Hegde Medical college, Nitte University, Mangalore, Karnataka, India.

²Department of Medicine, Manipal Hospital, Bangalore, Karnataka, India

⁴Department of Anatomy, Kannur Medical college, Kannur, Kerala, India

**Author for Correspondence*

ABSTRACT

The human immunodeficiency virus (HIV) infection gradually destroys the body immune system, which makes it harder for the body to fight infections. HIV infection causes a quantitative and qualitative depletion of CD4 lymphocyte count. With the progression of the disease there is a fall in CD4 lymphocyte count, which increases the risk of opportunistic infections. The present study was done in the department of Medicine, Manipal Hospital, Bangalore, Karnataka over a period of two years from February 2010 to February 2012. This study was done on 50 consecutive newly detected HIV positive patients suffering from opportunistic infections, who were admitted to the hospital. The present study, demonstrates the relationship between CD4 lymphocyte count and the occurrence of opportunistic infections in HIV infected patients and the type of opportunistic infections in HIV infected patients in South India. The data obtained were compared with the data of other studies.

Key Words: CD 4 count, HIV, AIDS, Opportunistic Infections.

INTRODUCTION

Human Immunodeficiency Virus (HIV) infection and the consequent acquired immunodeficiency syndrome (AIDS) has reached the status of a global pandemic, with cases being reported from virtually every country. The current estimate of the number of cases of HIV infection worldwide is approximately 34.2 million (UNAIDS, 2011). There were 2.5 million new infections and 3 million deaths during 2007 (UNAIDS, 2011). In India alone there were about 3.97 million people suffering from HIV infection. (Sharma, 2003). Two decades into the epidemic and there is still no cure or vaccine for AIDS. There is considerably more information now available on how the disease spreads, the immunopathogenesis and clinical course of the disease. This wealth of information helps in implementing prevention strategies and increased understanding about what constitutes effective management of HIV or AIDS patient.

It is the infectious complications of AIDS that first defined the syndrome beginning in 1981 when cases of clusters of Pneumocystis Jiroveci Pneumonia or Pneumocystis carinii Pneumonia (PCP) were first reported. Over the next several years, numerous other opportunistic infections were recognized including cytomegalovirus (CMV) retinitis, disseminated mycobacterium avium cellulare (MAC) infection, cryptococcal meningitis and toxoplasma encephalitis. Now that it is well into the second decade of HIV infection, great strides have been made in characterizing the various opportunistic infections that affect HIV infected patients and in refining our treatment approaches to these illnesses.

Fundamental in understanding AIDS related opportunistic infections is the appreciation of the relationship between the level of underlying immune dysfunction as measured clinically by the CD4 count drops below 250 to 200/ μ L. The main target of the HIV appears to be the CD4 cell population. A progressive reduction in the number and function of the CD4 cell population is one of the most striking and consistent immunological features of HIV-related disorders (Taylor *et al.*, 1989). The CD4 cell count is an important investigation in the clinical evaluation of any patient with HIV infection, as it helps to decide the stage of HIV infection. It also assists in differential diagnosis and in making therapeutic decisions regarding antiretroviral treatment and prophylaxis against opportunistic infections (Quinn, 1997). Yet certain HIV

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related infections (eg. bacterial pneumonia, tuberculosis, varicella zoster) can occur at any CD4 count although even these tend to predominate among patient with lower counts. Ample Western data showed the relationship between CD4 cell counts and the spectrum of opportunistic infections (Longo *et al.*, 2008 and Gerald, 2007) but very few Indian studies show the correlation between CD4 cell counts and the occurrence of opportunistic infections (Ghate *et al.*, 2000). HIV infected individuals in India are exposed to various environmental factors like malnutrition, poverty and a host of tropical infections which are peculiar to this region. Striking differences and similarities exist between clinical presentation of AIDS in Indian population and other countries (Sharma, 2003). With this knowledge, timely introduction of prophylaxis and therapeutic decisions can be made based on the CD4 cell counts.

MATERIALS AND METHODS

The present study was done on 50 newly detected HIV positive patients suffering from opportunistic infections who were admitted at the Department of Medicine, Manipal Hospital, Bangalore, Karnataka over a period of two years from August 2010 to August 2012. All patients were more than 16 years of age, were HIV infected and had opportunistic infections. The patients with pre existing immunocompromised status due to any cause other than HIV and/or patients on immunosuppressive therapy were excluded from the study.

Both the clinical and laboratory data were collected from the subjects after taking consent from the patients. Enzymes Linked Immuno Sorbent Assay (ELISA) was used for screening for HIV infection. Testing for antibodies to HIV was performed using the commercially available ELISA kit, which contains both HIV-1 and HIV-2. This test has 99.5% sensitivity. Patients who were tested positive were subjected to a second ELISA test on the same sample using a different kit. If positive, the result was termed 'repeatedly reactive'. Confirmation by Western Blot could not to be done due to non – availability of the test and financial constraints.

Immunophenotyping of lymphocytes was carried out by 2 colour analysis on FACSORT flow cytometer. The lymphocytes were stained according to the protocol suggested by the manufacturer. In brief, 100.µl of heparinized blood was mixed with 10µl of monoclonal antibodies [anti- CD3- FITC, anti- CD4-PE (BD)]. The red cells were lysed using solution (FACS lysin BD, Singapore) and after incubation for 30 minutes the cells were washed and fixed with formaldehyde – phosphate buffered saline (PBS). The cells were acquired on FACSORT and analysed in Simulset software (BD). Gating for lymphocytes was carried out by Leucogate reagent (CD45CD14 antibody BD, USA). Absolute CD4 counts were calculated by the following formula:
$$\text{CD4 count} = \frac{\text{Total leucocyte count} \times \text{lymphocytes \%}}{100} \times \text{CD4\%}$$

The clinical and microbiological diagnosis for Mycobacterium tuberculosis infection was diagnosed by detecting Acid fast bacillus (AFB) in smears, lymphocyte predominant body fluids, granulomas on biopsy, antimycobacterial antibody. PCP was diagnosed by clinical features, chest x-ray findings, arterial blood gas analysis showing partial pressure of oxygen (pO₂) less than 60mmHg and elevation of serum lactate dehydrogenase (LDH). Toxoplasmosis was diagnosed by computerised tomography (CT) scan of the brain and IgG Toxoplasma levels in serum. Cryptosporidiosis, Microsporidiosis and Isospora was diagnosed by stool examination. Fungal infections such as Cryptococcosis was diagnosed by staining with Indian ink, cryptococcal antigen and by biopsy. The Candida infection was diagnosed clinically and potassium hydroxide (KOH) stain. Histoplasmosis was diagnosed by biopsy. Viral infections like Herpes Simplex and Herpes Zoster was diagnosed by Tzanck smear. CMV infection was diagnosed clinically and by biopsy. Data thus obtained was statistically analyzed.

RESULTS

Majority of the patients were between the age group of 31 to 40 years with a mean age of occurrence of 41 years. The youngest patient in the study group was 16 years old male patient and the oldest patient was a 63 years old. The mean CD4 count was not affected by the age in our study group. Among 50 patients,

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80% of the patients were males and 10% were females. The mean CD4 counts were 196/ μ l and 246.4 / μ l for males and females respectively. 48 patients were married and 2 patients were unmarried. The mean CD4 count for married and unmarried patients were 193.27 / μ l and 196.50 / μ l. Table 1 shows the occupation of the HIV infected patients. 80% of the female patients were housewives. Table 2 shows the probable route of transmission of HIV to the patients. 70% of the patients gave a history of exposure to multiple sex partners. Among the female patients, 8% of them were diagnosed on the basis of their spouse being positive. 16% of the patients were not aware how they acquired disease. Most of the patients were from Bangalore (48%) and a significant number were from Bellary (08%), Andhra Pradesh (14%) and Tamil Nadu (06%). Table 3 shows the clinical presentation of the patient. The most common symptom was fever (96%), followed by weight loss (84%) and cough in 60% of patients. The most common clinical signs were pallor (42%) and lymphadenopathy (36%). Among 45 patients who underwent liver function tests, 42 patients had hypergammaglobulinemia.

Table 1: Occupation of the patient

OCCUPATION	NUMBER OF PATIENTS	
	MALES	FEMALES
Agriculture / unskilled	08	00
Employed / skilled	14	02
Business	14	00
Coolie	02	00
Driver	01	00
House wife	00	08
Others/student	01	00

Table 2: Route of Transmission

ROUTE OF TRANSMISSION	MALES	FEMALE
History of exposure to multiple sex partners	34	01
Spouse being HIV positive	00	04
History of Blood transfusion	01	02
Not known	05	03

Table 3: Percentage of clinical manifestations in present and previous study

Clinical Manifestations	Present study	Kothari et al.,
Fever	96%	96%
Weight loss	84 %	66%
Cough	60%	60%
Dyspnea	16%	
Abdominal pain	13%	6.6%
Diarrhea	11%	23%
Vomiting	12%	
Dysphagia	16%	30%
Altered sensorium	12%	26%
Neurological deficits	08%	10%
Pallor	42%	
Lymphadenopathy	36%	43%
Skin lesions	16%	33%
Genital lesions		23%
Oral ulcer		43%
Cyanosis		10%

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Table 4: Clinical features of HIV disease in developing countries

	Sub Saharan African,	Kenya	Latin America, Brazil	Asia, India	Thailand,	Present study
1. Number of HIV patients	349	95	111	3551	1553	50
2. Tuberculosis	28%	18%	32%	62%	37%	64%
3. Bacterial infections	18%	26%			<1%	16%
4. Isosporiasis	7%		6%		0	
5. Bacterial pneumonia	6%	16%	16%		<1%	16%
6. Cerebral Toxoplasma	6%		14%	3%	2%	6%
7. Bacterial enteritis	5%	6%				
8. Non specific diarrhoea	5%	15%				22%
9. Oesophageal candidiasis	3%		24%	57%	3%	6%
10. Cryptococcus	2%	1%	5%	4%	38%	2%
11. Kaposi's sarcoma	1%	2%	5%	<1%	<1%	
12. CMV	0		5%	1%	4%	

Table 5: Comparison of mean CD4 count in opportunistic infection between the present study and the Western studies

Infection	Present study n =50			Moore et al., n =1246			Crowe et al.,
	Case %	Mean CD4	Median CD4	Case %	Mean CD4	Median CD4	CD4 count
1. Tuberculosis	64	71.13	118	6	26	9	250-500
2. Oral Candidiasis	30	77.9	49				250-500
3. PCP	18	79.4	72	8.9	59	27	75-125
4. Cryptococcus	02	44		34	63	32	75-125
5. EsophagealCandidiasis	06	109.6	78	13.3	79	30	75-125
6. Herpes infection	12	293.5	289	5	195	107	75-125
7. Cryptosporidia	10	477	318	0.8	92	28	150- 200
8. Toxoplasma	06	76.6		0.8	44	22	75-125
9. CMV	04	52.5		6.9	37	15	50

Among patients infected with TB, 11 patients had Pulmonary tuberculosis and 8 patients had Disseminated tuberculosis. These two were the most common types of tuberculosis. (Table 6). Majority of the patients with tuberculosis in all groups had CD4 counts less than 200 (65.62%). The radiological manifestations in Tuberculosis are affected by the CD4 cell count. Atypical manifestation occurs when

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CD4 cell count are <200/ μ l and typical cavity and upper lobe infiltrates occur when CD4 counts are >200/ μ l.

Table 7 shows the relationship between CD4 lymphocyte count and opportunistic infection. The occurrence of Mycobacterium tuberculosis (TB) was 64%, it is evident from the table that the range for CD4 cell count in patient suffering from TB is wide with a median count of 118/ μ L. Oral candidiasis with the 2nd most common opportunistic infection (30%). 3 patients (6%) had esophageal candidiasis. The mean CD4 count for oesophageal candidiasis was higher (109.6/ μ L.) as compared to the mean CD4 count for oral candidiasis (77.9/ μ L). A presumptive diagnosis of PCP was made in 15 of the patient. The mean CD4 count was 79.4/ μ L. Among the 8 patient (16%) with pyogenic infections, 6 of them had bacterial pneumonia. One patient had urinary tract infection (E coli was isolated from urine culture).

Table 6: The relationship between CD4 count level and the type of Tuberculosis

TYPE OF TUBERCULOSIS	NUMBER	CD4 COUNTS			MEAN CD4	MEDIAN CD4
		<200	200-500	>500		
TB lymphadenitis	02		02		332	
Disseminated TB	08	05	03		194	246
TB meningitis/tuberculoma	05	04	01		114	48
Abdominal TB	05	04		01	201	113
TB pericardial effusion	01		01		305	
Pulmonary TB	11	08	03		125	102
Total	32	21	10	01		

Table 7: Frequency distribution of opportunistic infections in relation with the CD4 count

Type of infection		CD4 counts/ μ l					Total
		0-50	51-100	101-200	201-500	>500	
1.	Tuberculosis	07	06	08	10	01	32
2.	Oral candidiasis	08	02	04	01		15
3.	P.Jeroveci (PCP)	04	02	03			09
4.	Bacterial infection	02	02	02	02		08
5.	Diarrhea	01	02	03	04	01	11
6.	Cryptococcal infection	01					01
7.	Esophageal candidiasis	01	01	01			03
8.	Herpes infection			02	03	01	06
9.	Toxoplasmosis	02		01			03
10.	CMV infection	01	01				02
11.	Cryptosporidiosis	01		01	01	02	05
12.	Lymphoma	01	01				02
13.	Hepatitis				03	01	04

Table 8: Classification of the 50 cases in our study based on the CDC¹⁵ classification for HIV infections

CD4 counts	A	B	C
>500	A1=0	B1=01	C1=02
200-499	A2=0	B2=03	C2=09
<200	A3=0	B3=02	C3=33

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One patient had subacute bacterial peritonitis. The mean CD4 count for bacterial infections was 139/ μ L. 1 patient suffered from cryptococcal meningitis, diagnosed based on CSF findings. The CD4 count for cryptococci was 44 / μ L. Toxoplasma was diagnosed in three patients, on the basis of CT scan the brain. The mean CD4 count was 76.6 / μ L. Among the 16 patients suffering from diarrhea, cryptosporidia was isolated from stool of 5 cases with mean CD4 count of 477 / μ L. The remaining 11 patient had a negative stool examination and culture. 2 patients (4%) had CMV infection, both had IgG positive, one patient had CMV. Esophagitis diagnosed by biopsy. They had mean CD4 counts of 52.5 / μ L. Herpes infection was seen in 6 (12%) of the patient, of whom 1 had vaginal herpes (CD4 count of 143/ μ L). The mean CD4 count was 293.5/ μ L. 4 (8%) patients had hepatitis infection, of whom one had Hepatitis A and the remaining three patients had Hepatitis B. The mean CD4 count was 354 / μ L. 1 patient had progressive multifocal leukoencephalopathy and the CD4 count was 83 / μ L.

It is evident from Table 7 that the occurrence of opportunistic infections increased as the CD4 count dropped to less than 200/ μ L. The increased frequency was seen in all groups. Very few opportunistic infections occurred at CD4 counts of greater than 500/ μ L but when the proportion above and below 200/ μ L was compared; no statistical significance could be established for any of the diseases. 65% of the patient with TB had CD4 cell counts greater than CD4 cell count greater than 200/ μ L. But the comparison of number of TB below and above CD4 count of 200/ μ L, was not statistically significant. Among oral candidiasis also it was evidence that the range was wide, but the occurrence of the disease increased as CD4 cell counts dropped to less than 200/ μ L, (93.3% of the patient with oral candidiasis), but this was not statistically significant. All of the patients with PCP had CD4 counts of less than 200/ μ L. 75% of the cases with bacterial infection occurred below 200/ μ L as compared to 25% above the level. But this difference was not statistically significant. Similarly the occurrence of cryptococci, CMV and Toxoplasma infections were below 200/ μ L. These results could be because the sample size for some of these opportunistic infections was relatively small.

On classification of the 50 cases in our study based on the CDC (Centers for Disease Control), classification for HIV infections 88% patients fell into category C, 12% in category B, no patients in category A.

DISCUSSION

It is evident from the data of national AIDS surveillance program in India done in the month of December 2003 by UNAIDS, that the most common age of presentation of patients is between 30 to 44 years. A study done on 1246 HIV infected patients by Moore *et al.*, (1996) showed that 48% were in the age group 31 to 44 years. This is concordant with the present study where majority of patients were in the age group between 31 to 40 years. The number of patients presenting after the age of 45 years were fewer in both the present study and the other studies (Moore *et al.*, 1996). The youngest patient in the present study was a 16 year old male patient.

The mean CD4 count was not affected by the age of the patient in our study group. There was no correlation found between the age and CD4 count in our study and the study done by Ghate *et al.*, (2000). The mean CD4 count being much lower in the age group of 61 years and above compared to the 30 to 40 years age group, indicating that the disease was acquired at an early age. 80% of the patient were males and 20% were females, in our study, which was similar to the number as per the Indian surveillance UNAIDS. The National AIDS surveillance program clearly shows the number of male patients suffering from HIV disease is more (83.5%). And the study done by Ghate *et al.*, (2000) showed 76.8% were males and 23.1% females and by Moore *et al.*, (1996) showed 75% were males and 25% were females. The male patients were found to have a lower CD4 count than females in the present study and also in the study done by Ghate *et al.*, (2000) which can attributed to an early spread of the epidemic in men. 96% of the patients were married and 04% were unmarried, which is similar to the study done by Subhash *et al.*, (2003), where 80.5% of the patients were married. 40% of the patients belonged to the transport group Karnataka state AIDS prevention society (KAPS), 2002. But in the present study, no such major

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difference in number were noticed in the occurrence of the disease among drivers. There was equal distribution of patients among all the patients. Sexual mode of transmission was commonest mode of transmission of HIV infection in the present study (70%). This data is similar to data of National surveillance UNAIDS, which shows that sexual transmission was seen in 85.69%. The history of blood transfusion was present in 6% patients. 48% of the patients were from Bangalore, Karnataka. Being a referral center we had patients coming from Andhra Pradesh (14%), from Tamil Nadu (06%). A significant number (08%) of the patients were from Bellary. The surveillance done by the KAPS (2002) for the year 2002 show that the HIV epidemic is equally distributed throughout Karnataka and the prevalence of HIV exceeds more than 1% in antenatal clinic. The prevalence at Bellary is more than 2% in the antenatal clinic and 15 to 20% in the STD clinic. In India the prevalence of infection is 3% in Karnataka, 44.3% in Tamil Nadu and 10% in Andhra Pradesh (UNAIDS).

In our study, fever (86%), weight loss of more than 10% (81%) and cough (51%) were the most common presenting complaints. Anemia (42%) was the most common clinical sign followed by Lymphadenopathy (36%). This is similar to the study done by Kothari *et al.*, (2001).

It is a known fact that HIV infection causes hypergammaglobulinemia (globulins greater than 2.5mg/dl) due to B cell proliferation and immunoglobulin secretion which are abnormal in function Angelo *et al.*, (2004). This activated state manifests as hypergammaglobulinemia with a defective antigen response. In our study 93% of patients underwent liver function tests and all were found to have hypergammaglobulinemia

The results of our study demonstrate increased frequency and severity of the illnesses in HIV infected patients in association with depletion of CD4 count. The Table shows the occurrence of opportunistic infections in the developing countries and in India. It is evident that tuberculosis (TB) is the most common opportunistic infection in India and the present study. In our study TB was the most common opportunistic infection (64%) in our study. This occurrence was similar to other studies by Ghate *et al.*, (2000) and Subhash *et al.*, (2003), which showed the occurrence of TB as 29% and 25.4 respectively. Owing to the environmental conditions prevailing in India like malnutrition, poverty and over population the incidence of TB in the community is high as compared to other parts of the world. The incidence increases in patients with HIV infection, due to reinfection and relapses which can vary from 12% to 75% Kothari *et al.*, (2001). The occurrence of PCP, Cryptococcal meningitis, Toxoplasma and CMV infections were much less in our study and study in other parts India and the world as showed in Table 4. Oesophageal candidiasis was low in our study probably because of under diagnosis of the same. A higher number may have been diagnosed if endoscopy was done routinely for all patients with Oral Candidiasis.

The median CD4 count was low for Mycobacterium tuberculosis (118 / μ l) as compared to Western data, which indicate the median CD4 count for Mycobacterium tuberculosis as 356/ μ l (Gerald, 2008). But the range in our study was wide, 7 to 743/ μ l indicating that Mycobacterium tuberculosis occurs at any CD4 count, Tuberculous lymphadenitis at a higher CD4 level and disseminated tuberculosis at a lower CD4 level (table 6). From the table 3 it is evident that Mycobacterium tuberculosis had a lower incidence of 6% in the Moore *et al.*, (1996) study with the mean CD4 value of only 261 / μ l. But for patients with TB, in the Crowe *et al.*, (1991) study the reference range for CD4 counts were from 250 to 500/ μ l.

Candidiasis in our study, (both oral as well as oesophageal) occurred at a wide range of CD4 level; but the mean CD4 count (77.9 / μ l) for oral Candidiasis was significantly lower compared to the mean CD4 count (109.6/ μ l) for esophageal Candidiasis. Compared to our study, the Moore *et al.*, (1996) and Crowe *et al.*, (1991) studies showed that the mean CD4 count was much lower for esophageal candidiasis.

Data show that the mean CD4 count for primary PCP is 36/ μ l and secondaryPCP is 10/ μ l (Longo *et al.*, 2008). In our study the mean CD4 count was 107.21/ μ l which was similar to study done by Crowe *et al.*, (1991). But in the Moore *et al.*, (1996) study the mean CD4 counts for PCP was much lower (59/ μ l). The mean CD4 count for Toxoplasma, Crytosporidia and CMV in our study was very similar to the mean CD4 counts of Moore *et al.*, (1996) study. The Crowe *et al.*, (1991) study was one of the earlier studies, which gave a higher range of CD4 counts for each opportunistic infection than the values that were

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obtained in the present study. This disparity between the two studies could probably be due to the fact that, opportunistic infections may now be occurring only with severe immunosuppression.

Based on the AIDS case surveillance definition suggested by Centre for disease control and prevention (CDC) we observed that 88 % of the patients had severe disease (C is severe). Whereas the study done by Ghate *et al.*, (2000) observed that 33.6%, 47.4% and 19% had mild (A), moderate (B) and severe (C) respectively. This was explained in their study, by the fact that inadequate diagnostic facilities placed patients in category B, where they actually belonged to category C. The possibility of our study having a greater number of severe cases could be due to the fact that, our hospital being a tertiary care centre, only patients with advanced disease came for medical help.

It was also observed in our study that patients had multiple clinical conditions, this is based on the fact that persons with advanced HIV disease are more likely to suffer from more opportunistic infections due to greater immunodeficiency, reflected by low CD4 counts. But CD4 counts of a single clinical condition may be higher than the CD4 count for multiple clinical conditions. With all the above mentioned observations it is evident that the CD4 count is an adequate marker for progression of HIV infection and the occurrence of each opportunistic infection can be expected based on a single CD4 count value before the institution of prophylaxis or antiretroviral therapy.

But it has been reported that it may be difficult to use CD4 counts as a surrogate marker for HIV infections/ AIDS in India because variations in CD4 counts due to factors such as ethnicity, low body mass index (BMI), associated malabsorption, tuberculosis and tropical diseases can individually affect the CD4 counts Angelo DM *et al.*, (2004).

Such data needs to be validated and more detailed studies are required to explain such occurrences. It has also been proposed that diurnal variation in CD4 count can occur. We have minimized such affects by collecting fasting samples. To overcome such controversies serial CD4 counts needs to be done to corroborate the association between CD4 counts and opportunistic infections. But in a country like India getting even a single value is a great financial burden to the patient.

Specific diseases occur below certain CD4 counts, except in conditions where the occurrence of the disease is high, like tuberculosis and oral candidiasis. In our study there were patients referred from other states for management of HIV who were in the advanced stage of the disease, which could explain the wide spectrum, severity and low CD4 counts for each infections. Serial declining CD4 values may have a better implication in the degree of immunosuppression than a single value, due to non -HIV infection mediated fall in CD4 counts. Larger community based studies need to be done and followed up for several years to get a better picture of the relationship between opportunistic infections and CD4 counts. This will help in better interpretation and applications of CD4 count for setting guidelines for possible points of intervention and prophylactic algorithms for each opportunistic infection.

Conclusion

Sexual exposure was the major route of transmission. The mean CD4 count was much lower in the age group of 51 to 60 years, possibly indicating that the disease was acquired early with severe immunosuppression at time of presentation. No relation to profession of the patient was found. The most common clinical presentation was fever, weight loss and cough. The most common clinical findings were pallor and lymphadenopathy. Hypergammaglobulinemia was seen in majority (93%) of the patients. Tuberculosis (TB) was the most common opportunistic infection (64%), followed by Oral Candidiasis (30%), Pneumocystis Jiroveci Pneumonia (PCP) (18%), Bacterial infections (16%), Herpes infection (12%), Cryptosporidia (10%). Pulmonary TB was the most common type of TB. The CD4 count range in patients with TB was very wide ranging from 23/ μ l to 609/ μ l, but a significant number of patients had CD4 count greater than 200/ μ l. This wide range of occurrence could be due to reactivation and relapse of TB seen commonly in India. The mean CD4 count for TB lymphadenitis was much higher as compared to disseminated TB and abdominal TB. The difference between the mean CD4 count for pulmonary and

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disseminated TB was not statistically significant. The radiological manifestations of TB were more atypical when the CD4 counts dropped to less than 200/ μ L.

Oral candidiasis had a wide range of CD4 counts from 4 to 700/ μ L. The mean CD4 count in our study for Oesophageal Candidiasis was much higher than for oral Candidiasis. This can probably due to under diagnosis of Oesophageal Candidiasis. The mean CD4 count for each of the disease Pneumocystis jiroveci pneumonia (PCP), Cryptococci, Toxoplasma, CMV and for Cryptosporidia of the present study were similar to those of Western studies.

The incidence of the opportunistic infections increased as the CD4 count dropped below 200/ μ L. But this difference was not statistically significant, as the sample size was too small for each disease. In conclusion, the present study showed that mean CD4 count for opportunistic infections occurring in India were comparable to the CD4 counts for opportunistic infection given in Western literature. However in case of TB and Candidiasis where the occurrence in the present study was high, no particular CD4 count can be taken as a reference value.

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